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INTRODUCTION

The intent of this handbook is to inform producers, veterinarians and extension personnel regarding some of the basics of controlling internal parasites that affect sheep and goats in Canada. It is not meant to replace the unique relationship between the veterinarian and the producer and his / her flock or herd, but to augment the information available.

This handbook was written primarily for those working with sheep and goat farms in Ontario as well as Canada, where research over the past decade carried out by several research institutions has helped us to understand the epidemiology of internal parasites in this country. However, this information should also prove useful to those living in other parts of the world - particularly those with a similar temperate climate. This handbook will always be a work in revision, as we better understand parasitism of sheep and goats in this country. At the time of writing of this version, considerable research is ongoing in Canada on parasite immunity, genomics and developing new tests to detect resistant parasites. We hope that you will find it of use in developing internal parasite control programs for sheep flocks and goat herds.

IMPORTANT GASTROINTESTINAL NEMATODE PARASITES

This section contains a description of the most commonly found and clinically important gastrointestinal nematode (GIN) parasites of sheep and goats found in Canada. Information on other important but less common nematode parasites of the gastrointestinal tract, lung and nervous system, as well as other internal parasites are to be found towards the end of the handbook. Unless indicated, the life cycles of the GIN are similar as described in Figure 1. In Canada, the most common and the most pathogenic (disease causing) nematode parasites of sheep and goats are *Teladorsagia circumcincta*, *Trichostrongylus* spp. and *Haemonchus contortus*.

HAEMONCHUS CONTORTUS

WHAT DOES IT LOOK LIKE?

This very common parasite is also called the **"barber pole worm"**, **"blood worm"**, or **"wire worm"**. The worms are large (1.5 to 3.0 cm), easily visible to the naked eye and the female oviduct is visible as a white stripe around the red blood-filled intestine, giving it a barber-pole appearance. It is found on the surface of the abomasum (glandular or 4th stomach). *H. contortus* eggs are oval and medium sized (65-100 X 35-50 µm) - typical of the Trichostrongyloidia Superfamily and is indistinguishable from eggs of many of the GIN parasites discussed here and are often called GIN eggs.

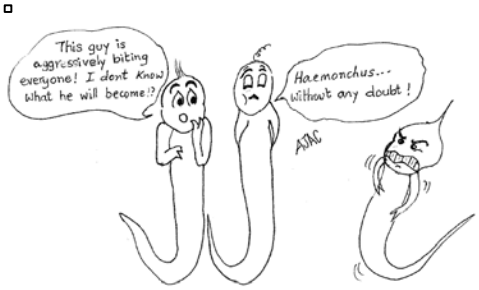
Trichostrongyle-type egg

HOW DOES IT LIVE?

This most pathogenic parasite of sheep and goats also infects llamas, deer and occasionally cattle. *Haemonchus* is a tropical parasite but has been very successful in establishing itself in cold climates such as Canada. However, the infective third-stage larvae (L3) do not survive well over winter on pasture when temperatures drop below 0° C. Although a few L3 may be found in the spring in the pasture grasses from the previous grazing season, they are weak and are not very successful at establishing infection in an animal.

However, it is a very prolific egg producer - each female worm can produce 5,000 to 10,000 eggs per day - enabling it to rapidly increase the pasture contamination to dangerous levels by mid-July. Pasture contamination comes from two sources: infected lambs and kids are the main contributors, but early contamination comes from parasites that have overwintered in adults; these produce large numbers of eggs at lambing/kidding. It is important to manage both sources of pasture contamination.

The eggs can develop to the infective L3 stage in as little as 4-5 days (see Figure 1) but require fairly warm temperatures to do so. Egg development may be delayed up to 2 months if the weather is cool, although the eggs are sensitive to environmental conditions. The L3 larvae can survive for months on pasture under moist conditions.

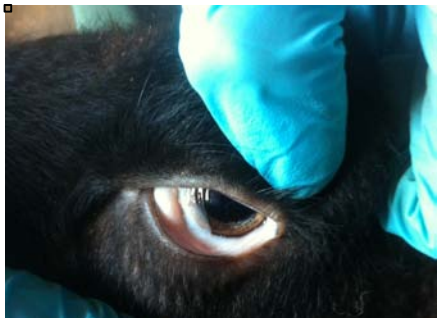


Severe disease outbreaks (haemonchosis) usually occur mid-July to August in young-stock as well as adults on pasture but the exact time depends on the air temperature (i.e. prefers > 25° C) and moisture. So outbreaks occur earlier if the summer is hot and wet, and later in cool summers. Hot

and dry conditions are not favourable to survival of L3 on pasture. Finally, many of the ingested L3 larvae become "arrested" in the abomasum, starting in early fall and do not complete development until the following spring around lambing-time (periparturient).

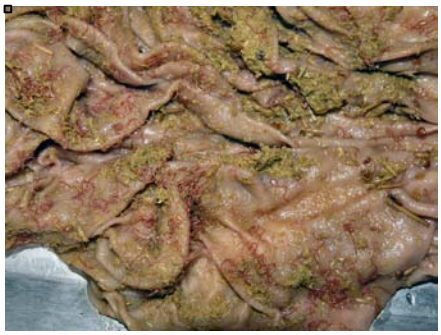
DISEASE IN THE SHEEP AND GOAT

The adult worm has a lancet mouthpiece that enables it to pierce the mucosal surface of the abomasum. It then feeds on the blood that seeps from the wound. One worm can result in the loss of 0.05 ml blood per day from ingestion and seepage from the wounds they make. A load of 5,000 worms will cause a loss of 250 ml (1 cup) daily. A 25 kg lamb or kid (55 lb) only has 2000 ml of blood in total. However, clinical disease can occur with a load as few as 500 worms. In the course of a few weeks, infected animals will become severely anaemic from this blood loss. The bone marrow tries to compensate by increasing red blood cell production, but it is often a losing battle. The animal may drop dead on pasture with very severe infections (e.g. 30,000 worms). This is the classical presentation of disease and is sometimes termed "**Type I haemonchosis**". Animals with lower levels of infection will be chronically anaemic, have low protein (hypoproteinemia) and "bottle jaw", and have a poor appetite and weight loss. The mucous membranes around the eye (conjunctiva) appear pale pink to white. The haematocrit (the proportion of blood that contains red blood cells) is often less than 12%, indicating severe anaemia (normal 25% - 35%).



Anaemic and hypoproteinemic lamb; *courtesy C. Schwartzentruber*

In Canada and northern countries, "**Type II haemonchosis**" often occurs in the spring, even before the animals go to pasture. Ewes or does may develop severe disease usually around lambing / kidding or when nursing. The large number of parasites that were picked up while grazing the previous season and overwintered as larvae in their abomasum, emerge as adults and start to feed in the spring.



Haemonchus contortus parasites in the abomasum; *courtesy N. Sargison, University of Edinburgh*

DIAGNOSIS

Egg counts in the feces tend to be very high when correlated with the number of adult parasites in the abomasum; counts of 3,000 - 5,000 eggs per gram are not unusual with worm counts of 500. However, 500 worms can kill a lamb so don't disregard lower egg counts. Additionally, you cannot tell which GIN parasite the eggs belong to so it is prudent to interpret counts > 1,000 eggs per gram as possibly being dangerous to the animal.

On postmortem, the carcass is very pale due to the anaemia. In cases of low protein, the tissues under the skin may appear watery. The abomasum contains numerous visible worms (500 to 20,000). The contents are dark brown from the seeping vessels and excreted digested blood, which may make it difficult to see the worms. Floating the abomasal contents in a dish of water will help to see the worms. In severe, acute infections the abomasal wall (mucosa) may appear swollen and haemorrhagic.

TELADORSAGIA CIRCUMCINCTA

WHAT DOES IT LOOK LIKE?

It is a small parasite, found in the abomasum, approximately 1 cm long, and barely visible to the naked eye. Also known as the "**brown stomach worm**" and formerly known as "**ostertagia**". Eggs are typical GIN eggs and are indistinguishable from *Haemonchus* eggs.

HOW DOES IT LIVE?

This parasite infects sheep and goats. It burrows into the glands of the abomasum that produce digestive juices, where it then feeds. The burrowing may destroy these glands. Most severe infections occur in the late summer or fall but in rare circumstances, severe disease is associated with the re-emergence of the overwintering larvae in the spring (**Type II Teladorsagiosis**). The infective third-stage larvae (L3) are well adapted to survive over winter on pasture in the Canadian climate and do so very successfully! A pasture that was severely contaminated the previous autumn will still be severely contaminated in the spring when animals are turned out to graze. However, because the parasite produces fewer eggs than *Haemonchus*, it may take longer to badly contaminate a new pasture in our northern climate – this may change as our climate becomes hotter. The arrested stage carried in ewes and does over the winter will develop in the spring and contribute significantly to pasture contamination with the periparturient rise in egg output.

DISEASE IN THE SHEEP / GOAT

Disease is most often seen in lambs and kids during the first season on pasture. Infection is associated with intermittent diarrhea, weight loss or reduced gains, and decreased appetite. Plasma pepsinogen (a digestive enzyme in the abomasum) levels may be elevated due to abomasal damage, and the pH of the abomasum is increased (normal is 2.0 - 2.5) because of damage to the glandular cells that secrete hydrochloric acid. This interferes with digestion and contributes to diarrhea, ill thrift or weight loss. The inflammation from the infection also makes the animal feel ill, furthering a drop in appetite. A load of 5,000 worms is considered to cause significant clinical disease. It is possible for severe disease to occur prior to eggs appearing in the feces; lambs or kids put to heavily contaminated pastures may experience severe disease due to the sudden massive infection.

Wall of the abomasum of an adult goat. The nodules indicate inflammation and scarring due to infection with *Teladorsagia*. This goat was very thin and had diarrhea.

DIAGNOSIS

Egg counts of > 500 eggs per gram may indicate an important infection, as this parasite is less prolific in producing eggs. On postmortem, typical damage is seen. The parasites invade glands in the mucosa of the abomasum and cause swelling and redness of the abomasal folds. Scarring will occur with loss of gastric function - sometimes permanently in severe infections. If scarring is severe, the animal will not get better after deworming – even though the parasites are killed.

TRICHOSTRONGYLUS AXEI

WHAT DOES IT LOOK LIKE?

This parasite is also known as "**stomach hairworm**". It is also an abomasal parasite. The worms are < 0.5 cm in length and very difficult to see with the naked eye. Eggs are typical GIN eggs and not distinguishable from *Haemonchus* or *Teladorsagia*.

HOW DOES IT LIVE?

It is a parasite of sheep and goats but also infects cattle and deer. The adults burrow into the wall of the abomasum and feed. Like *Teladorsagia circumcincta*, disease is usually seen in the late summer or fall after a build-up of L3 on pasture. The L3 are well adapted to survive over winter on pasture in the Canadian climate. The arrested stage in sheep/goats will develop in the spring and contribute significantly to pasture contamination with the periparturient spring rise in egg output.

DISEASE IN THE SHEEP / GOAT

Diarrhea, hypoproteinemia (bottle jaw), decreased appetite, weight loss are again all clinical features of severe infections with this parasite. Elevated plasma pepsinogen and abomasal pH are also important features as with *Teladorsagia*. A load of 5,000 worms is considered to be associated with clinical disease.

DIAGNOSIS

As with Teladorsagia, egg counts of > 500 eggs per gram could be reflective of significant infection. The parasites invade the glandular mucosa, causing damage to the secretory cells. On postmortem, raised plaques may be visible on the abomasal surface with chronic infections.

TRICHOSTRONGYLUS COLUBRIFORMIS AND *T. VITRINUS*

WHAT DOES IT LOOK LIKE?

Also known as the **"black scour"** or **"bankrupt"** worms, they are small (0.5 to 0.75 cm in length), light brown and hair-like worms found in the small intestine (duodenum and upper jejunum). Eggs are typical GIN eggs.

HOW DO THEY LIVE?

These parasites infect sheep, goats and cattle (*T. colubriformis* only). The larvae burrow into the mucosa of the small intestine to develop and then burst out about 10 days after infection. This may cause severe damage to the intestinal wall with loss of blood and protein. Most disease occurs in the late summer and fall from the build-up of pasture contamination. L3 can over-winter on pasture very well and serve to infect lambs and kids in the spring grazing. They can also overwinter in the animals as arrested L3 parasites. While trichostrongyles can cause significant disease alone, the worst disease outbreaks are usually seen with co-infections with *Teladorsagia*.

DISEASE IN THE SHEEP / GOAT

The parasite causes a dark diarrhea and hypoproteinemia (bottle jaw) with poor appetite and weight loss. Milder infections are associated with soft stools and poor growth rates. Affected lambs and kids may have manure (dag) sticking to the back end and tail – evidence of diarrhea.

DIAGNOSIS

Egg counts are similar to Teladorsagia with significant infections. On postmortem, the small intestine will have patches of erosion and loss of the normal villous lining visible on microscope.

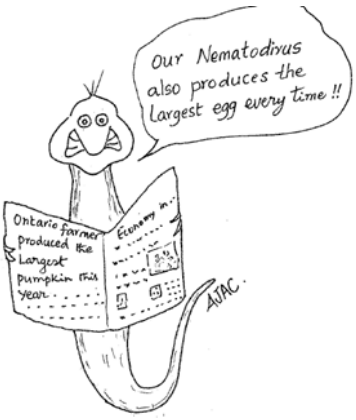
NEMATODIRUS

WHAT DOES IT LOOK LIKE?

Also known as the **"thread-necked worm"**. They are slender worms approximately 1 to 1.25 cm in length and live in the upper part of the small intestine. The front part of the worm is more slender than the rest of the parasite. With a heavy infection, they appear like a cotton-ball in the upper small intestine. The worms produce very large eggs inside which the larvae develop to the L3 stage. They are much larger than the eggs of other GIN parasites (152-260 x 67-120 µm). The eggs of *N. battus* are orange-brown in colour whereas *N. filicollis* and *N. spathiger* are colourless, so they can be differentiated under the microscope. These parasites infect sheep, goats and occasionally calves.

HOW DO THEY LIVE?

N. battus is the most disease-causing species of this genus. In contrast, *N. filicollis* and *N. spathiger* cause only mild or no disease. Their life cycle is very different from other GIN parasites. With *N. battus*, the eggs will only hatch after a prolonged period of cold weather followed by more mild weather in which the temperature stays above 10° C. Usually, eggs laid in the summer do not hatch until the following spring or possibly even for 2 years, so that the biggest risk period for infection and disease is the late spring (May and June). Lambs and kids on pasture in the spring are most at risk of disease. However lambs and kids housed indoors or in dry lots can become infected indicating that the parasite can complete its life cycle without pasturing. Adult sheep and goats appear to have very good immunity and don't seem to play a role in infecting their offspring.



DISEASE IN SHEEP AND GOATS

N. battus will cause severe watery yellow-green diarrhea in lambs and kids, often accompanied by dehydration and thirst - and in severe infections, death. Clinical signs may appear before eggs are produced (prepatent period of 14 to 16 days), so faecal egg counts may be of limited value in the face of clinical disease. Mild infections of *N. filicollis* and *N. spathiger* may have no to mild signs of disease -most infections are seen with other GIN parasites.

DIAGNOSIS

A small number of eggs usually represent a large number of parasites in the animal. Large numbers of thread-like "cotton balls" of worms will be found in the small intestine. Severe infections are indicated by mild reddening of the intestine (enteritis) and marked atrophy of the intestinal villi (the folds of cells lining the small intestine responsible for absorption of nutrients).

THE TYPICAL LIFE CYCLE OF A GASTROINTESTINAL NEMATODE PARASITE

Figure 1

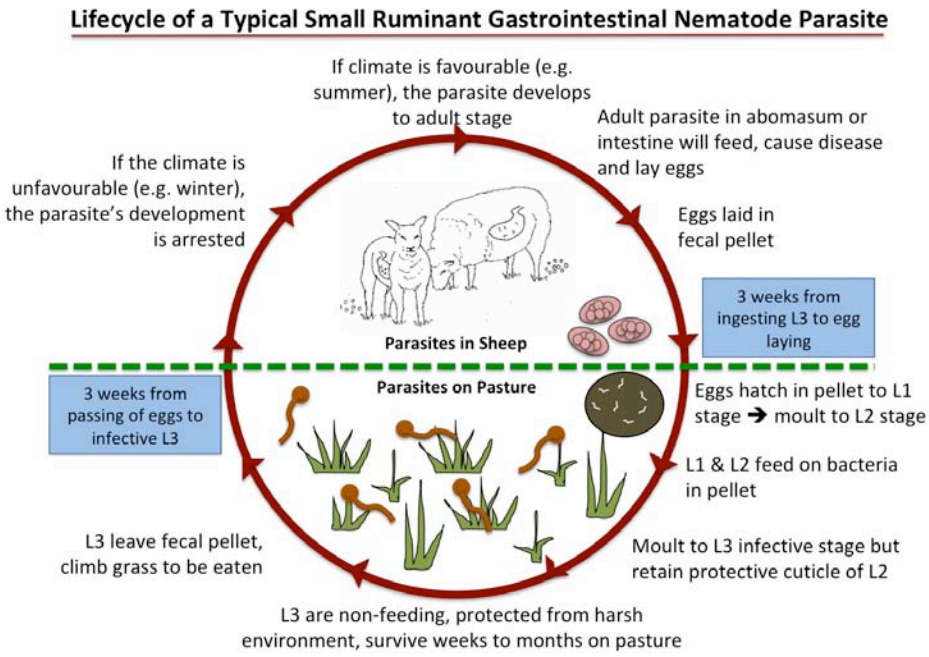


Figure 1 shows the typical life cycle of the gastrointestinal nematodes *Teladorsagia circumcincta*, *Haemonchus contortus* and *Trichostrongylus* spp. For these parasites, there is no intermediate host. The **Prepatent Period** is the period from ingestion of the L3 stage to when eggs are detected in feces, usually 16 to 21 days for most GIN.

- The eggs of the parasite are passed in the feces.
- The eggs hatch within the faecal pellet to release L1 larvae.
- L1 larvae (free-living) feed on bacteria within the pellet and moult to L2.
- L2 larvae (free-living) feed on bacteria within the pellet and moult to L3.
- L3 larvae retain the L2 cuticle. This is the infective stage to the sheep / goat. This stage does not feed and relies on stored nutrients for survival. This stage migrates outside the faecal

pellet on pasture, is capable of climbing several centimetres up a grass stalk, and is ingested by the sheep / goat when grazing.

- The time from egg expulsion to L3 varies depending on the weather and species from 5 days to 6 weeks or more.
- Inside the gastrointestinal tract the L3 moults to L4.
- L4 larvae are parasitic and feed on the sheep / goat and moult to immature and then mature adults or…
- L4 become arrested (hypobiotic), and do not feed or develop to adults until environmental conditions are more favourable.
- The adult stage, which is parasitic, lays eggs that are passed in the feces.
- The cycle starts over again.

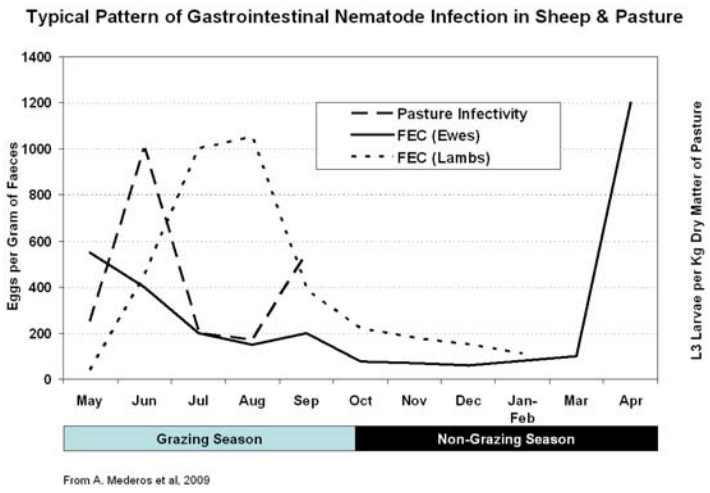
EPIDEMIOLOGY OF GASTROINTESTINAL PARASITES

We will examine how the parasite interacts with its host (the sheep or goat) and the environment and what determines how well the parasite survives and whether disease occurs in its hosts.

Figure 2 summarizes gastrointestinal nematode parasite burden in lambs and adults, as well as the number of infective L3 on pasture, under conditions in central Canada. This graph is adapted from data obtained on farms in Ontario and Quebec in 2006 - 2008. The abstract of this published work is included in Appendix 5. In the spring, adult sheep/goats serve to contaminate the pasture for youngstock, which then are the major source of pasture contamination through the grazing season. A hot dry summer will lower pasture contamination, while numbers will rise with the warm rains that often occur in late August - September.

NORMAL PATTERNS OF INFECTION IN ADULTS AND YOUNGSTOCK

Figure 2



YOUNGSTOCK IN THEIR FIRST GRAZING SEASON

Lambs and kids have no acquired immunity to gastrointestinal nematode (GIN) parasites, although some may have an innate (born with) immunity of varying levels. The L3 stage on pasture infects naive lambs and kids. The level of L3 on pasture, the level of immunity in the youngstock will determine the level of disease seen. Over the grazing season, the parasite load in the lambs and kids tend to increase and they quickly become the major contributors to egg contamination on pasture. Towards the end of the grazing season, a proportion of the ingested L3 will not progress to adults, but will rather become hypobiotic or arrested in development. If youngstock are not grazed, then they will not develop immunity until they are grazed as adults (e.g. yearlings). Immunity in sheep takes several months (up to 5 months) to develop. Goats do not develop immunity well.

ADULT SHEEP AND GOATS

Adults, if they have been previously grazed, may carry infection over the winter as arrested L4, and will contribute to pasture contamination when turned out to graze in the spring from the emerging adult parasites. The level of egg shedding will depend on the parasite exposure and level of immunity they developed in the previous grazing season. Adult goats develop immunity less well than sheep and may have a more important role than adult sheep in contaminating pasture. The phenomenon of periparturient (around lambing / kidding) egg rise is due to a lowering of immunity (called "down-regulation") around the time of lambing or kidding (see below). This allows for increased egg production by parasites, and thus serves to be one of the most important sources of pasture contamination in the spring to lambs and kids.

HYPOBIOSIS OR ARRESTED LARVAL DEVELOPMENT

After the L3 larvae infect the host and moult to the L4 stage (for Trichostrongylus – L3) in the animal, they may either develop into adults or arrest as larvae. At this stage, little disease is seen in the host and no eggs are passed. In this way, many immature parasites may collect in the host without clinical disease. The trigger for hypobiosis is thought to be unfavourable environmental conditions for egg hatching and development of the free-living larval stages, e.g. the cooling weather of autumn in temperate climates or the dry season in the tropics. In Canada, arrested development is an important mechanism that allows for survival of *H. contortus*, as well as *Teladorsagia* sp. and *Trichostrongylus* spp. It is believed that in Canada most L3 larvae ingested in the fall - and late summer in the case of *Haemonchus* - arrest rather than develop to adults.

IMMUNITY AND PARASITE BURDEN

ACQUIRED IMMUNITY TO PARASITES

Lambs and kids (though less well) will develop immunity to parasites over time. The actual length of time varies with the type of GIN but generally occurs over the first grazing season if long enough (4-6 months). Nursing lambs and kids tend to have no immunity but also have very little exposure to GIN parasites until they start to graze. Once they graze, immunity – at least in lambs – starts to develop. However, the time for immunity to develop in an individual varies between species or breeds of animals and between animals in a flock. There is a moderate heritability of this ability, which scientists are working to identify and exploit with specific genomics testing and breeding programs. When immunity develops, the adult parasites are expelled but the animal will continue to be infected with low numbers. This is called "**self-cure**".

Acquired immunity to parasites can be lowered by several factors. It is known that immunity to parasites is short-lived. Without continued exposure to parasites, the animal's immunity will wane and after a few months, it can become susceptible again. We see this in Canada when sheep / goats in the spring will have poor immunity after being housed all winter. Additionally, challenge with high numbers of GIN on pasture can overwhelm the animal's immune system and cause disease.

Immunity is also greatly affected by nutrition. Diets deficient in protein, particularly dietary protein as rumen non-degradable protein, also called "by-pass" protein, can lower the animal's ability to mount an immune response to GIN parasites. This type of protein is not digested by the rumen bacteria, but passes through the rumen and is digested in the abomasum and intestine. Examples of this type of protein are fishmeal, roasted soybeans and corn gluten. Supplementation with diets with higher protein is associated with lower egg counts in sheep; this is most easily seen at lambing and lactation. Some pasture plants containing condensed tannins (e.g. sanfoin and bird's-foot trefoil) can also supply this type of protein. It is very important to remember that goats do not develop immunity as well as sheep.

PERIPARTURIENT EGG RISE (PPER)

This phenomenon is due to the "periparturient relaxation of immunity" (PPRI), and refers to the increase in eggs passed in the feces of ewes and does from a few weeks before giving birth through the nursing period (around 8 weeks). This typically takes place in the spring months and occurs because of a down-regulation (lowering) of immunity in the late-pregnant female which allows for the following: arrested larvae to mature to egg-producing adults; ingestion of overwintered L3 on pasture to more likely result in infection; and an increased rate of egg production from existing adult worms. All of this results in a dramatic increase in pasture contamination in the spring at lambing / kidding and while nursing. Nutritional stresses in late pregnancy and lactation will increase the likelihood of PPER being high. PPER tends to be lower in single-bearing females compared to those with multiples, lower in mature females than first-timers, and lower when females are supplemented with by-pass protein sources.

PPER in out-of-season lambing flocks

Findings from research conducted in Ontario flocks on accelerated lambing programs found that ewes lambing in the winter also experience a PPER, while those lambing in the fall already had very high levels of infection and PPER was not apparent. The abstract describing this work is presented in Appendix 5. Thus PPER will occur regardless of season although it appears to be less of a feature in fall lambing ewes, perhaps because the parasites have not yet become hypobiotic. Canadian data also suggest that PPER may be extended in dairy ewes. Perhaps this is due to a higher level of nutritional stress.

GENETIC RESISTANCE TO INFECTION WITH GIN PARASITES

Immunity to parasite infections may be innate (i.e. the animal is born with the ability to manage parasite infections) or acquired after exposure to parasites. Both are important. Some sheep and goats develop better immunity against parasites, and are more able to resist establishment. This can be more pronounced in some breeds, e.g. some tropical hair breeds of sheep are known to develop immunity more quickly than some northern wool breeds. Also, within any population of sheep or goats, there is variation in this ability, and a portion of that ability is genetic. This trait is considered moderately heritable.

Programs have been developed to identify sheep that carry this genetic ability, either through ram selection (e.g. rams raised together and selecting those with lower faecal egg counts), or attempting to identify genetic markers in the DNA. Both of these methods have drawbacks:

- There is tremendous variation in day-to-day faecal egg counts (FEC) and so to use FECs, a large number of animals are needed to detect significant differences in egg output that are due to genetics.
- Additionally, the animals need to be exposed to significant burdens of parasites to have high enough FEC to detect a difference between animals.
- Gene markers may detect animals of one breed with parasite resistance but in another breed, these markers don't work.

Newer methods of measuring immunity instead (e.g. CarLA Saliva Test, AgResearch NZ) have shown promise. Fewer animals are needed to detect differences and lambs with superior performance can be detected as little as 3 months after going to pasture. Research is being conducted in Ontario to determine if this technology can be used here.

It must be remembered that lambs or kids with superior ability to develop acquired immunity, are susceptible to GIN infection until that immunity is developed and so need to be monitored for infection. The benefit comes later, after they have an opportunity to develop immunity. As adults, these resistant animals will shed fewer eggs, most importantly at the time of PPER, or if faced with an excessive burden on pasture. Their offspring are also more likely to be able to manage parasite challenges.

RESILIENCE TO INFECTION WITH PARASITES

This is the animal's ability to thrive in the face of parasitic infection. These animals are infected, and pass eggs that contaminate pasture, but appear to be healthy. Within any population, there are individuals that exhibit resilience. But they serve to contaminate the pasture for animals that are not resilient or immune. It is also less heritable than the trait for superior immunity. This means that while some animals aren't affected by increased pasture contamination, a larger part of the flock is affected and may suffer disease. So for this reason, resistance is preferred to resilience.

NORMAL PATTERNS OF INFECTIVITY ON PASTURE

EFFECT OF ENVIRONMENT ON DEVELOPMENT AND SURVIVAL OF THE FREE-LIVING STAGES

Temperature

At temperatures < 10° C, egg hatching, larval development and moulting do not occur. Parasite larvae develop at temperatures above this but prefer warmer temperatures for optimal development. The optimal temperature for hatching, larval development and L3 survival varies by parasite:

- *Teladorsagia circumcincta* prefers 16° - 30° C;
- *Trichostrongylus colubriformis*, 22° - 33° C; and
- *Haemonchus contortus* prefers the warmest temperatures at 25° to 37° C.

Northern spring weather conditions may not be favourable to rapid egg hatching and larval development. But when temperatures range from 25° to 30° C during the summer, the development of all parasite larvae is favoured. When summer weather is hot, while the other parasites usually take 3 weeks from egg to L3, *Haemonchus* may develop in as little as 5 days. **As our climate continues to warm, all GIN parasites – but in particular *Haemonchus* – will enjoy more favourable conditions for development.**

Although warm weather hastens parasite development to L3, it may conversely lower the length of survival of those larvae. When the weather is hot (e.g. > 28° C), the L3 may die more rapidly because their metabolic rate increases and they outlive their stored nutrients (remember L3 cannot feed).

Conversely cool weather, which slows larval development, will also significantly increase survival of L3 from weeks to months because of the lowered metabolic rate. At temperatures < 5° C, the metabolic rate of L3 is very low - allowing prolonged survival, e.g. over-wintering on pasture.

Humidity

At faecal pellet or ground level, the humidity should be > 80% to allow for development. L3, but not L1 or L2 can survive desiccation (drying) because of protection of the cuticle covering, even at freezing temperatures.

ASSUMPTIONS REGARDING DEVELOPMENT AND SURVIVAL OF FREE-LIVING STAGES ON PASTURE

Survival of L3 over-winter on pasture

Some species of L3 (e.g. *Teladorsagia* and *Trichostrongylus* but not *Haemonchus*) can enter a state of "anhydrobiosis" that allows them to survive severe cold and desiccation (drying), making them well suited for surviving the freeze-thaw cycles of our Canadian winters.

When sheep or goats are turned out in the spring to a pasture that has been grazed the previous late summer or autumn, it can be assumed that it is contaminated with L3 that have survived over-winter. If pasture contamination was high the previous autumn, then the level of L3 from *Teladorsagia* and *Trichostrongylus* will be high in the spring as well. Snow cover throughout the winter enhances survival. Several freeze-thaw cycles or prolonged, cold temperatures without snow cover may lower this survival rate. *Haemonchus* does not survive effectively when temperatures dip below 0° C. Over-wintered L3 of all GIN are considered to survive no longer than the end of June - but this depends on the temperature and humidity. A cool, wet spring may enhance survival, whereas a warm / dry spring will shorten survival.

Development and survival of L3 on pasture during the grazing season

As mentioned, the time from egg deposition to development to L3 varies depending on temperature and humidity. Moderate temperatures and high humidity hasten development to L3. Moderate temperatures will prolong survival of L3. It is likely that a proportion of L3 present in June and July, are still alive in September. Hot temperatures will shorten survival. The pasture itself will influence development and survival. Old pastures with a mat of dead grasses above the soil, will hold humidity longer as well as reduce temperature extremes, and therefore enhance development and survival. Heavy grazing (e.g. through pasture rotation) will reduce this mat and open the soil to sunlight and desiccation - both limiting survival. Heavy morning dews or moisture that may be present after a rainfall, will allow migration of L3 a few centimetres up the grass blades, enhancing infectivity of the pasture. Hot, sunny days will drive the L3 down to the soil level, thus reducing infectivity.

Generations of GIN on pasture

One generation is the time from when the eggs are passed in the feces, through hatching and development of the free-living larvae, infection of the sheep or goat and then passage of eggs again in the feces. Assuming that time from egg deposition to L3 is 2 to 3 weeks (up to 6 weeks if cool) and time from infection to egg production is 3 weeks – so a total of 5 to 6 weeks on average. It is likely that optimal environmental temperatures are only present for 4 months in most of Canada (mid-May, June, July, August, mid-September ~ 16 weeks), so there is limited time for build-up of L3 on pasture during the first grazing season on a given pasture. This may change as our climate warms.

Teladorsagia and *Trichostrongylus*

In central Canada, it is unlikely that more than 3 generations of these parasites occur under our summer conditions. Therefore, it is likely that if severe parasitism from these parasites occurs on a farm, there was heavy contamination from the previous grazing season as well as high stocking densities in the current grazing season - along with optimal summer conditions for L3 development and survival, i.e. warm and moist conditions.

Haemonchus

Although *Haemonchus* doesn't survive on Canadian pasture over the winter, it survives very well in adult sheep and goats in the hypobiotic (arrested) state. Once the adult female worm matures in the spring, it is capable of producing 5,000 to 10,000 eggs per day. Under warm, humid conditions L3 will develop in as little as 4-5 days, allowing for multiple generations per grazing season (one generation could be as short as 3 ½ weeks) and therefore massive pasture contamination. This means that within one grazing season, the infectivity of the pasture may become very high and therefore risky to lambs and kids – and even adults - by mid-July to early August.

SURVIVAL OF L3 INDOORS

Very little is known about survival and transmission of GIN indoors. There is sufficient anecdotal evidence to suggest that infection can occur in the barn. Certainly lambs and kids housed in drylot situations (i.e. where they have access to a yard without pasture), particularly if fed on the ground – may pick up GIN infection. Inside a barn, in the summer it may be possible for eggs to hatch and develop to L3, but again – lambs and kids would need to be fed on the ground to consume sufficient larvae to cause a significant infection. Water bowls and troughs contaminated with feces are another source of infection. Very rarely are parasite burdens acquired indoors of clinical significance. To lower the risk of GIN in this situation, it is important to minimize manure contamination of feed and water.

DIAGNOSING GASTROINTESTINAL PARASITISM

FECAL EGG COUNTS (FEC)

FECs are a measure of the adult parasite population in the sheep or goat, but not a measure of total infection (i.e. L4 and immature adults). There is considerable animal-to-animal variation in FEC, so it is important to sample a random proportion of the group in order to get an accurate picture of the parasite load of that group.

WHO TO SAMPLE

Sheep or goats grazing pasture that are representative of the group should be sampled. Do not sample animals that have been held off feed for any reason, or that are off-feed due to illness. Ideally submit 10 samples representative of lambs/kids and 10 samples representative of ewes/does. It is important to sample youngstock separately from adults, as counts will be very different - even on the same pasture.

GETTING THE SAMPLES

The simplest way is to group the animals into a corner of the pasture (with clean ground), hold them for 15 minutes and then release them. Pick up 10 individual faecal samples that are fresh (warm), ideally not trampled, and place in individual plastic bags, e.g. sandwich bags. Immediately remove as much air as possible and place in a cooler with ice packs. Faecal samples can be collected from the rectum if you choose. Again, use a plastic bag or disposable glove. Apply a small amount of lubricant (e.g. methylcellulose or KY® Jelly) to the gloved finger and gently tease out the faecal pellets. This latter method allows results to be tied to an individual animal if required and not later pooled. For pooled results, animals must be randomly selected. See **Appendix 1** for more detailed information on how to collect, label and store samples.

TRANSPORTATION AND STORAGE OF THE SAMPLES

It is very important that the samples be kept cool (< 5°C) but not frozen until they reach the laboratory. This is to prevent hatching of the eggs, which will lead to underestimation of the level of parasitism. Refrigerated samples should be analysed within 7 days of collection.

ANALYSIS OF THE SAMPLES

A trained person should examine the samples to prevent confusion with air bubbles, plant cellulose and pollen or other artefacts commonly found in feces. It is also important that the samples be evaluated using a quantitative technique. The modified McMaster technique is one such method that will allow the number of eggs per gram (epg) of feces to be reported. Qualitative counts (e.g. 1+, 2+, 3+) are not useful for differentiating between a moderate infection (e.g. 150 epg) or a severe infection (e.g. 1,500 epg) as both will be interpreted as 3+. See **Appendix 2** for a description on how it is performed.

POOLED VERSUS INDIVIDUAL SAMPLES

There is considerable animal-to-animal variation in egg output, with 30% of animals responsible for ~ 70% of the egg output. Pooling of samples, so that only one test is done per group of animals is a valid way of analysing parasite load. However, samples should be pooled at the laboratory (not at the farm) to make sure that an equal weight of faeces is contributed by each animal (minimum 4 grams of feces each). For this reason, it is important to collect individual samples in individual bags when submitting. While results from individual samples will allow the veterinarian to identify and treat the most severely parasitized, it is much more expensive to run 10 individual samples rather than one pooled sample.

WHAT ARE SIGNIFICANT EGGS PER GRAM LEVELS?

The following cut-points are often used for individual or pooled samples:

- Low = <500 epg;
- Moderate =500 to 1,000 epg;
- Severe =>1,000 epg.

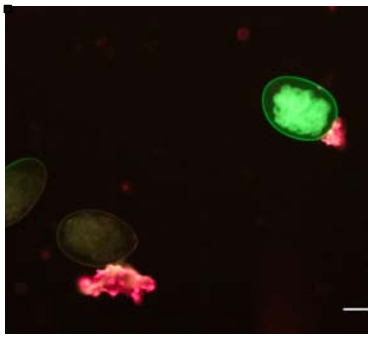
However, there are several factors that need to be appreciated when deciding what cut-point to use or what action to take:

Species of GIN

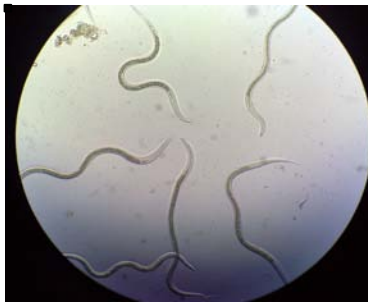
Haemonchus is a very prolific egg producer and is associated with rapid changes in pasture infectivity. Moderate infections with *Haemonchus* may have FEC of > 1,500 epg – a level that would signal a severe parasite load with any other GIN species. If *Haemonchus* is the predominant GIN, the FEC can change very quickly, as can the level of disease in the lambs/kids. Furthermore, even within the 3-week prepatent period of the parasite, youngstock can become very anaemic - before egg levels change significantly.

Generally, we do not know which type of parasite is contributing to the GIN FEC but can use history of previous infections to help. A technique utilizing fluorescent peanut agglutination (PNA) lectin binding is used in some commercial laboratories to stain and identify the proportion of eggs that are of the *Haemonchus* type.

Larval culture and identification of GIN species can be done in specialized laboratories, although that service is not routinely provided in central Canadian diagnostic laboratories. The standard technique involves hatching and culturing the eggs to the L3 stage. Identification of the different species involves morphological differences including measuring tail length – a very laborious and possibly inaccurate procedure. A new technique being developed by the University of Calgary involves culturing the eggs only to L1 stage (much more easily done) and using a PCR method (detects parasite DNA) to differentiate species. Preliminary work at that University along with collaboration with the University of Guelph, has demonstrated that this method is very accurate.



Florescent PNA Lectin staining to identify *Haemonchus* parasite eggs (upper right). *Courtesy Chris Pinard and Trisha Westers*



Cultured L3 larvae. *Courtesy J. Avula*

Emergence in the spring of arrested larvae from infection from the previous season

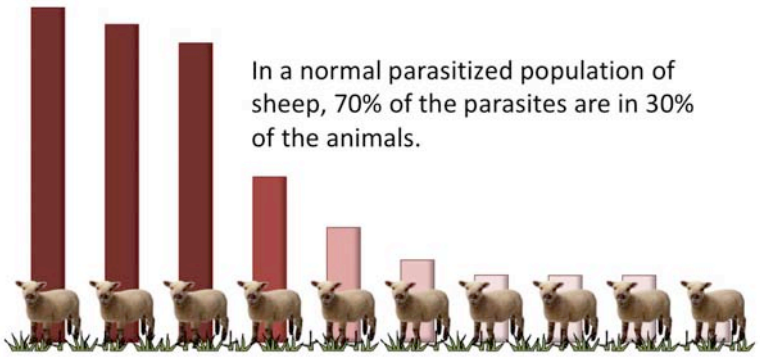
In the winter months, sheep/goats that grazed the previous summer may have a significant hypobiotic (arrested) load of GINs that are sitting in or on the abomasal wall waiting for more favourable climate conditions before developing to adults. In the spring, the re-emergence of large numbers of these larvae can be associated with significant disease - often called "Type II" disease. The animals have diarrhea (*Teladorsagia*), bottle jaw and / or anaemia (*Haemonchus*) along with a negative FEC as the larvae have not yet reached the adult egg-producing stage. Understanding when this might happen in a flock or herd is critical to proper interpretation of FECs.

Grazing heavily contaminated pastures

Naive animals that graze very heavily contaminated pastures may experience disease due to *Teladorsagia* and *Trichostrongylus* before the prepatent period is complete. Like Type II disease, these animals will have watery diarrhea and bottle jaw with some deaths - along with a very low FEC.

Individual variability in FEC

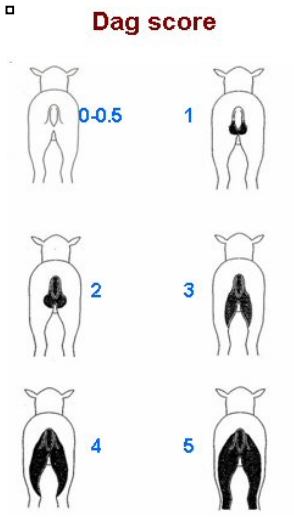
It has been shown that approximately 30% of young-stock are responsible for 70% of the total egg production. This means that there is tremendous animal-to-animal variation in egg output - also called "over-dispersion" by researchers. If averages are used to determine how infected a group of animals is, there is a great risk of underestimating the level of infection. An example: 3 faecal samples have a count of 1,000 epg and 6 faecal samples have a value of 50 epg, this gives an average value of 367 epg. In this example, if a cut-point of 500 epg is used, it might result in a decision not to treat when treatment should have been performed on the high-shedding animals. Factors that should also be taken into account include the clinical condition of the animals, as outlined below.



CLINICAL CHANGES IN THE ANIMAL

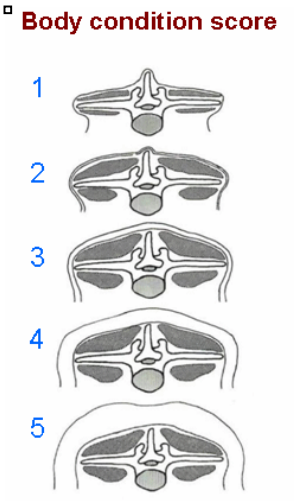
DIARRHEA / DAG SCORES

Faecal consistency (formed pellets, soft pellets, liquid diarrhea) may reflect parasite load, but some animals with parasite infections (e.g. acute haemonchosis) do not exhibit diarrhea. An animal with coccidiosis may have severe diarrhea but no GIN. Diet type also greatly influences faecal consistency, with lush grass causing diarrhea, so interpreting dag score must be done in light of the type of pasture being grazed. Dag is defined as faecal contamination of the wool or hair coat around the tail and hindquarters. Soft or diarrheic stools will cling to the wool / hair. A dag score will give an approximation of faecal consistency or prevalence of diarrhea in the group of sheep/goats. It should be noted that animals with diarrhea might actually have decreased FEC because the eggs are diluted, so low FEC in an animal with diarrhea, does not always mean that animal is not parasitized.



POOR WEIGHT GAINS / WEIGHT LOSS

Gastrointestinal parasitism is associated with poor growth rates. The poor growth is primarily due to decreased appetite from the parasite infection. Additional factors are the energy losses associated with the animal fighting the infection (i.e. immune response) and the losses of protein and blood that the parasites consume. Producers that weigh lambs/kids on pasture, can track growth rates and use this information (along with FEC) to determine if parasitism is clinically important. This



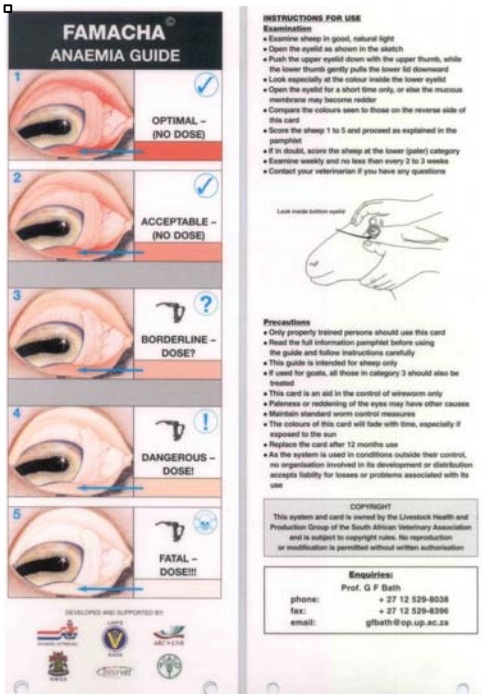
may be one of the most sensitive indicators of significant levels of parasitism in individual sheep/goats. A weigh scale set-up so that it is easy to run lambs/kids through every few weeks during the highest risk periods, can allow for selective treatment of those animals not gaining as expected. At the same time, the level of anaemia can be assessed (FAMACHA© score, see below). However, there are other causes of poor weight gains (e.g. poor pasture, coccidiosis, pneumonia) so that FEC should be done to confirm a parasitism problem.

Body condition score (BCS) is difficult to use as a reliable indicator of parasite burden, as so many other factors influence BCS. By the time an animal is thin (≤ 2.0), it is also experiencing severe clinical illness – too late for a screening test to be of good use. For these reasons BCS is not the best indicator to use to determine level of parasitism. However, an animal in poor BCS may be more susceptible to parasites particularly if debilitated from another disease such as Johne's disease (paratuberculosis).

ANAEMIA (*HAEMONCHUS*)

A major clinical feature of haemonchosis is anaemia. In central Canada, from late July on in warm and wet summers, haemonchosis is often the most important type of parasitism on some farms. Lambs and kids can be monitored during this period for evidence of anaemia. This can be done by taking a blood sample and measuring the proportion of red blood cells (packed cell volume or haematocrit). But assessing the level of anaemia can also be done by assessing the colour of the conjunctival mucous membrane. This is the tissue inside the eyelids. The colour is normally pink but it can be pale pink to white in significant *Haemonchus* infections.

The FAMACHA© system makes use of this. A card with different colours of pink (from very pink to white) is compared to the conjunctivae of the lower eyelid of the animal and the level of anaemia is estimated. It was developed in South Africa in regions where the primary type of GIN is *Haemonchus*, and is used successfully in many parts of the world where this parasite is common. It allows the producer to monitor individual animals and to only treat those that appear anaemic. Its drawback is that if other parasites are important, then it will fail to detect those infections so faecal egg counts must always be done when using this tool. There are other parasites that can cause anaemia, coccidia and liver flukes being two. It is also very labour intensive and requires good handling facilities that allow easy restraint of the head. **Every sheep / goat should be scored at least every 3 weeks and every 2 weeks when *Haemonchus* is a problem in the flock.** Colour should be assessed ideally in the daylight. If this is not possible, barn light or a non-LED flashlight is suitable. LED flashlights have been shown to wash-out colour leading to the conclusion that the animal is more anaemic than it is in reality.



FAMACHA card; *courtesy of G. Bath, University of Pretoria, South Africa*

FAMACHA© scoring and interpretation

Animals that score 1 and 2 (pink) are considered healthy and do not require treatment. Animals that score 4 and 5 are considered severely anaemic and should be treated immediately with an effective dewormer. Animals that score 3 should be watched carefully and dewormed if there are > 10% of the group scoring 4 or 5.

The scoring is subjective, however and mistakes do occur. It is possible that animals are scored 4 or 5 and are not anaemic and don't require treatment. This error has little consequence for the animal, as its health is not at risk if it is treated when it isn't necessary. But animals may be scored 1, 2 or 3 when they are actually severely anaemic. Not being treated when they should be is considered a fatal error; i.e. the animal may die because of misclassification. The system is designed to minimize fatal errors, but proper use of the FAMACHA© card and interpretation of findings requires training before using. The FAMACHA© system should only be used under the guidance of a veterinarian and as an adjunct to routine FEC. If you believe that the FAMACHA© system would be useful on your property, contact your veterinarian for training on how best to use it. On-line training is also

available at the University of Rhode Island Northeast Small Ruminant Parasite Control website: https://web.uri.edu/sheepngoat/famacha/

HYPOPROTEINEMIA (BOTTLE JAW)

Almost all the GIN parasites feed on protein, also called albumin, which circulates in the blood and lymphatic system. Bottle jaw in Canada is most commonly associated with *Haemonchus* infections. In severe infections, the protein levels can drop very low and the fluid, which normally stays in the blood and lymphatic vessels, leaks out and gathers under the skin and in the gastrointestinal lining. When fluid accumulates under the jaw, this is termed "bottle jaw". Edema in the gastrointestinal lining causes diarrhea and poor absorption of nutrients. By the time this is clinically apparent, parasitism is very advanced and the animal is in danger of dying.

Bottle jaw due to haemonchosis

POSTMORTEM EXAMINATION AND WORM COUNTS

If animals are dying and internal parasites are suspected of being the cause, it is very important to confirm this diagnosis with a total (adult) worm count from the gastrointestinal tract. Do not assume every dead lamb or kid found at pasture died due to worms - demonstrate this with a postmortem performed by a trained veterinarian.

A veterinarian can perform a field postmortem and attempt to identify abomasal and intestinal nematodes. *Haemonchus* are large and easy to see. *Teladorsagia* and *Trichostrongylus* are small and should be counted in the laboratory using a microscope. The abomasal contents are removed and volume measured; a known volume is then removed and the worms counted. For example, if there are 20 worms counted in 1/10th of the volume of the fluid in the abomasum, then the abomasum contained 200 worms. A system recommended for interpreting burdens, in the manual for the Sustainable Control of Parasites in Sheep (SCOPS, 4th edition) from the UK, is as follows:

2 points = Parasitism is likely affecting productivity
3 points = Parasitism is likely causing clinical signs and even death

Effects are additive

Teladorsagia spp:	3000 worms	= 1 point
Trichostrongylus spp:	4000 worms	= 1 point
Haemonchus contortus:	500 worms	= 1 point
Nematodirus spp:	4000 worms	= 1 point
Immature worms:	4000 worms	= 1 point

ANTHELMINTIC DRUGS FOR SHEEP AND GOATS

Currently in Canada, there are three anthelmintic drugs (dewormers) licensed for use in sheep, specifically ivermectin drench and injectable (e.g. Ivomec®, Merial Canada Inc.), closantel drench (Flukiver™, Elanco Canada Limited) and a combination drench containing derquantel and abamectin (Startect™, Zoetis Canada Limited). There are no anthelmintics licensed for use in goats in Canada at this time.

Veterinarians licensed by the province in which they practice have the ability to prescribe anthelmintics that are licensed for use in other livestock species. But with this ability comes the responsibility for assuring safety (to both people and animals), efficacy (must actually work against the parasites) and appropriate withdrawal times for meat and milk. Your flock veterinarian can obtain scientifically valid meat and milk withdrawals by contacting CgFARAD (https://cgfarad.usask.ca/language.php) prior to dispensing the anthelmintic for use. Please be aware that when a drug is not licensed for use in a particular species (e.g. goats), there is no established maximum residue limit (MRL) of that drug allowed in meat or milk. This means that if an anthelmintic is detected in (e.g.) goat milk, even if the level is lower than that allowed for cow's milk (for example), this is a violable residue and the milk will be discarded and the owner possibly fined. So just because it is OK to use in dairy cows, doesn't mean it is OK to use in dairy sheep or dairy goats.

Anthelmintics are divided into broad spectrum - i.e. those able to kill a wide variety of parasites, and narrow spectrum - those only able to kill one or two types of parasites. The following is a short discussion on how the various drugs work and their range of activity.

BROAD-SPECTRUM ANTHELMINTICS

MACROCYCLIC LACTONES (ML)

This group contains the avermectins (ivermectin, doramectin, eprinomectin) and the milbemycins (moxidectin). These compounds are derived from specific species of the *Streptomyces* bacteria and all work similarly. MLs have activity against most nematodes including the L4 stage, but not tapeworms or flukes. The MLs block the transmission of electrical activity in the nerves and muscles of the parasite, causing paralysis. The mechanism is such that they do not pose a risk to mammals. They are not ovicidal, i.e. do not kill eggs passed by the parasite. They also have activity against some arthropod ectoparasites, specifically sucking lice and nose bots (*Oestrus ovis*), as well as some activity against keds (*Melophagus ovinus*) and mange (*Chorioptes*, *Sarcoptes* and *Psoroptes*). Because of this spectrum of activity, drugs in this class are sometimes called endectocides.

When administered, the drugs are stored in fat tissue and then slowly released into the body. These pharmacokinetic properties result in long meat and milk withdrawal times (ivermectin = 15 days meat withdrawal for drench and 35 days for injectable). Moxidectin is considered to have significant prolonged activity - approximately 35 days when administered as an injection, and 21 days when administered as a drench, against *Teladorsagia* and *Haemonchus*. Neither form is available in Canada and cannot be imported under the new regulations for "Personal importation of certain drugs for food-producing animals" as they do not meet the requirements of being a "List B" drug[1].

Eprinomectin pour-on (Eprinex® Multi 5 mg/ml pour-on for beef and dairy cattle, sheep and goats, Merial Animal Health Limited) is licensed for sheep and goats in the United Kingdom for adult GIN parasites (not immature and not external parasites) but is not in Canada so its use is considered extra-label drug use (ELDU). Use of pour-on products in small ruminants is generally discouraged because absorption may be different. With this product, the wool / hair must be parted on the backline and the nozzle placed against the skin, to make sure the product contacts the skin directly. If your veterinarian chooses to use eprinomectin, the topical dose is twice that of cattle (1 mg/kg bw). Your veterinarian should contact CgFARAD to determine the appropriate meat / milk withdrawal for small ruminants when used at this dosage.

Spiroindole – ML combination

Recently a new combination anthelmintic product was developed and licensed for use in sheep in Canada, Startect™ (Zoetis Canada Ltd.). The product contains both derquantel, the first drug of the novel spiroindole class and abamectin, an ML in the same class as ivermectin. It is the first combination anthelmintic available in Canada for ruminants. It is not licensed and should not be used in goats, as there is no safety or efficacy data for that species.

Derquantel acts as an antagonist at the nicotinic cholinergic receptors; it blocks contraction of the somatic muscle of the parasite resulting in flaccid paralysis. When combined with abamectin, which also causes paralysis of the parasite but by a different method, the product is very effective at killing a wide range of internal nematode parasites including some inhibited stages of those parasites, but not flukes or tapeworms. It has also been demonstrated to be effective against GIN parasites that are resistant to other anthelmintics, including MLs. At the point of writing, resistance of parasites to this combination has not been demonstrated anywhere in the world.

BENZIMIDAZOLES (BZ)

These are also known as "white" drenches. This group of chemicals is effective against all GIN and somewhat against adult tapeworms. The drugs are deposited in the rumen and are slowly released into the gastrointestinal tract. They act on the intestinal cells of the nematode and the skin cells of the tapeworms, inhibiting polymerisation of microtubules. This, in turn, inhibits uptake of glucose and causes starvation. They kill not only the adult forms but also the immature stages (L4). They are also ovicidal - with activity against eggs being passed by nematodes and tapeworms.

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1 https://www.canada.ca/en/public-health/services/antibiotic-antimicrobial-resistance/animals/personal-importation-certain-drugs-food-producing-animals.html#a3

Currently, fenbendazole is the chemical most commonly used from this group for sheep (Safeguard® 10% suspension, Merck Animal Health) followed by albendazole (Valbazen®, Zoetis Canada Inc.). Both of these products are available as drenches and are licensed for cattle but not sheep or goats in Canada. Albendazole also has activity against adult flukes, but should not be used during breeding or the first 45 days of pregnancy because of toxicity to the foetus in early pregnancy. Generally, however, the BZ class of drugs are very safe with low levels of toxicity. The dosage used in sheep is the same as the cattle dosage but goats metabolize the drug quickly and require the dosage to be doubled.

IMIDAZOTHIAZOLES (LV) AND TETRAHYDROPYRIMIDINES

This group contains levamisole, pyrantel and morantel. They are also known as "yellow" drenches. Levamisole is no longer licensed in Canada (since 2005) but is still in use in other countries. It is also used as an immunomodulator in humans and for the treatment of specific types of cancer. The difference between the dose that is toxic to animals versus the dose that is efficacious against GIN, is very narrow making overdose and poisoning a risk. Prior to this drug being taken off the market, the most commonly reported adverse drug reaction in any animal was from the use of levamisole in goats. Because of the narrow safety margin, its use is not recommended. It also cannot be imported under the new regulations for "Personal importation of certain drugs for food-producing animals" as it doesn't meet the requirements of being a "List B" drug.

Levamisole works by paralysing the parasite so that it is removed rapidly from the gut. It works well against a broad range of adult worms but less so against the immature stages (e.g. L4). However, it is particularly effective against lungworm. Signs of toxicity in animals include salivation, slow heart rate and muscle tremors with occasional death. Morantel can be used to treat GIN but is not effective against the immature forms. Pyrantel is rarely used in livestock.

AMINO-ACETONITRILE DERIVATIVES (AAD)

The first product from this new class of drugs (monepantel) was released March 31, 2009 in New Zealand and the UK (Zolvix®, Elanco Animal Health) but not in Canada. This was the first new class of anthelmintics developed in 25 years and has excellent activity against resistant strains of GIN as well as immature forms of nematodes, and in particular *Haemonchus*. The drug is also of low toxicity as it targets a unique, nematode-specific class of acetylcholine receptor subunits. Unfortunately, since its release there have been many reports of resistance in small ruminant GIN. This reminds us how important it is to use these drugs properly. Zolvix cannot be imported for own use as it doesn't meet the criteria of a "List B" drug.

NARROW-SPECTRUM ANTHELMINTICS

These drugs only act against a few types of parasites.

CLOSANTEL

This product was recently licensed in Canada for the control of *Haemonchus contortus* (Flukiver™, Elanco Animal Health). The drug is effective only against internal parasites that ingest blood. In sheep and goats, this is *Haemonchus* (including larval stages) and the liver fluke *Fasciola hepatica*. It acts by inhibiting ATP synthesis in the parasite's mitochondria affecting its energy metabolism. It also has persistent activity by strongly binding to the host's plasma proteins (albumin) so is delivered directly to the parasite ingesting the blood. Closantel is very persistent but not active against the non-feeding immature stages of *H. contortus*.

PRAZIQUANTEL

This drug acts against the adult and immature stages of tapeworms and is of most use for control of tapeworms in guardian and working dogs. It is not available for use in sheep and goats.

ROUTE OF ADMINISTRATION

DRENCH VERSUS INJECTION

Drenches are deposited in the rumen so that proper absorption can occur. Injection of anthelmintics has been shown to result in a longer action - which may be favourable in some instances, but may select for resistant nematodes because

of a prolonged period when the drug is only present at sub-therapeutic levels. **Therefore, use of injectable anthelmintics (i.e. ivermectin) is strongly discouraged.**

USE OF POUR-ON ANTHELMINTICS

There is evidence that pour-on products are not as well absorbed in sheep and goats as in cattle. Because of the risk of sub-therapeutic dosing by this route, they are **not recommended** for use in either sheep or goats. For more information on eprinomectin, please read above on MLs.

USING AN ANTHELMINTIC BY A ROUTE OTHER THAN INDICATED ON THE LABEL

Use of pour-on products as an oral medication is not recommended as the absorption, efficacy, duration of action and withdrawal times cannot be predicted. This increases the risk of the parasite developing resistance to the drug. Additionally, use of injectable products as a drench may not be advisable as they have a different carrier that can affect a drug's effectiveness.

APPROPRIATE DOSAGE OF AN ANTHELMINTIC

In Table 1 is a listing of those anthelmintics that may be available for use in sheep and goats in Canada. The dosages provided are based on Canadian labels or – if not licensed in Canada, licensed recommendations from other countries where the drug is approved, or from the literature. Please note, anthelmintics not approved for that species **should only be used on the advice of a licensed veterinarian and with a valid veterinary-client-patient relationship.**

Table 1. Suggested dosages of anthelmintics for treatment of GIN infections (bw = body weight)

	Benzimidazoles	Avermectin	Closantel	Derquantel/Abamectin
Sheep	5 mg/kg bw	0.2 mg/kg bw	10 mg/kg bw	0.2 mL/kg bw
Goat	10 mg/kg bw	0.3 mg/kg bw	10 mg/kg bw	DO NOT USE

Very Important: Please note that all dosages listed above (except derquantel / abamectin) are based on the amount of the active ingredient to be given per kilogram of body weight of the animal. Various formulations of the drugs have different concentrations of the active ingredient, and so the actual volume of drug delivered to the animal must be calculated based on the dosage, the concentration of the drug, and the weight of the animal.

EFFICACY AGAINST…

Table 2. Activity of anthelmintics against the different parasite classes

	Benzimidazoles	Avermectin	Closantel	Derquantel / Abamectin
Hypobiotic Larvae	+	++	+/-	++
Persistent Activity	-	+/-	++	+
Tapeworms	+	-	-	-
External Parasites	-	+	+	+
Liver Flukes	+/- *	-	++	-

+ = good activity; ++ = much activity; - = no activity; +/- = slight activity against most GIN parasites
* = albendazole has activity against adult flukes but only at double-dosage (10 mg/kg bw sheep)
Benzimidazole = fenbendazole and albendazole
Avermectin = ivermectin, doramectin, eprinomectin
Closantel in goats may have less persistent activity than in sheep

WHEN TREATING WITH AN ANTHELMINTIC DOES NOT WORK

When a treatment fails to clear up a problem with internal parasites, i.e. clinical signs of parasitism are still present and / or faecal egg counts are still high, this may be because of anthelmintic resistance or because of how the dewormer was selected and administered. It is important to understand both reasons so this can be corrected.

DRENCH FAILURE

This is the term used when the treatment fails to work. It is important to firstly investigate reasons other than anthelmintic resistance.

USE OF THE WRONG ANTHELMINTIC

If an anthelmintic is used to treat a parasite for which it has no efficacy, then treatment failure will occur. A common example is the use of ivermectin to treat tapeworms. Closantel is only effective against *Haemonchus*.

FAILURE TO ADMINISTER AN ANTHELMINTIC PROPERLY

There are many reasons why an anthelmintic is administered in such a way that it will not work. It is the responsibility of the producer and veterinarian to make sure that the risk of this happening is minimized. The following is how to prevent drench failure.

Weigh the animals to be treated

Use a calibrated livestock scale to prevent under-dosing (underestimating weight). If the animals are variable in weight, dose for the heaviest in the group. If the group has extreme variability in size (e.g. youngstock and adults together), separate into two groups and dose to the heaviest weight in each group.

Use only drugs that have a Canadian Drug Identification Number (DIN)

Drugs obtained through the Internet (for example) may not contain what the label says it does as they may be manufactured in countries that do not have strict legislation on quality control. A DIN indicates that the drug was manufactured under Canadian regulations and rules. Changes to the own use importation rules limit imports of anthelmintics to Canada. Products licensed in Canada but also licensed in the USA can be imported for own use but the product should have evidence of FDA approval on the label.

Select the correct dosage

Read the label for products approved for sheep. If not labelled for sheep or if using in goats, obtain the correct dosage by veterinary prescription. Table 1 has some suggested dosages.

Proper dosage for goats

Goats often require a higher dosage than sheep and cattle, sometimes twice as much. This is because goats will metabolize (use up) anthelmintics faster than sheep and cattle, so the drug disappears faster from the body and doesn't get a chance to do its job. Before using, get advice from your veterinarian. Additionally, withdrawal issues for meat and milk must be considered.

Doubling the dose versus treating twice

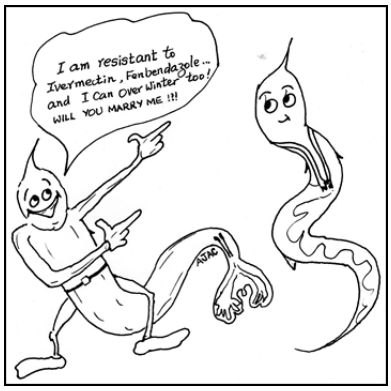
Doubling the dose provided in Table 1 will not double the effectiveness of the drug. Some drugs can be toxic if the dose is doubled, particularly if the animal is ill with parasites. But if necessary because of anthelmintic resistance (see below), repeat the dose 12 hours apart with BZ and ML drugs, as this will lengthen the time in the GI tract when an effective level of drug is present.

Calibrate your drench gun frequently

To assure delivery of the correct volume of dewormer and thus dose, it is important to frequently check the drench gun to make sure it is delivering the amount that is indicated. Drench guns frequently do not actually deliver the amount listed on the syringe so measure and compare. To calibrate your gun, draw up the drench and then "inject" 2 doses into a syringe (take out the plunger and put your finger over the end). This will accurately measure what you just delivered. Adjust the drench gun so it delivers the correct dose.

Drench correctly

This is done by firstly making sure the animal's head is properly restrained. The gun can do damage to the back of the throat if it jumps and fights while being treated. Such agitation can also result in the animal not being properly treated. Do not lift the head too high, as that will prevent proper swallowing. Gently insert the tip of the gun into the side of the mouth where there is a gap in the teeth, and direct it over the tongue, at the back of the throat. This will ensure that the drench is swallowed into the rumen and is more slowly released. If administered in the front of the mouth, loss may occur by spitting or having the drug swallowed directly into the abomasum where it will pass through the digestive tract, quickly reducing its effectiveness.



An excellent video description of how to drench is located at: https://www.wikihow.com/Drench-Sheep

If injection is performed

Make sure that the automatic syringe is calibrated appropriately and that the entire dose is injected subcutaneously (not "intra-wool").

Don't use an incorrect route of administration

Do not use a cattle pour-on product either as a pour-on or as a drench. Do not use an injectable product orally. Do not use pour-on deworming products as a pour-on as they may not be absorbed adequately to be effective and may contribute to development of anthelmintic resistance.

Hold animals off feed prior to treatment

Holding the animals off-feed for 12 hours before treatment will increase the length of time that the anthelmintic is effective. This is only effective if using BZ drugs, and should not be done if the ewes/does are in late gestation because of the risk of pregnancy toxaemia.

REINFECTION AFTER TREATMENT – APPARENT TREATMENT FAILURE

If the pasture that the animals are turned out to graze after treatment is infected with high levels of L3, then there can be apparent treatment failure. Many anthelmintics have no persistency; very soon after treatment the lambs/kids are re-infected from the L3 on pasture. If the challenge from contaminated pasture is high, then clinically they may appear as if they have not responded to the treatment. Depending on when the faecal samples are re-examined (e.g. 2 weeks later), the FEC may be very low indicating that the parasites within the animals were killed - however, immature adults may be numerous enough to cause disease. Reducing the challenge post-treatment through pasture management prevents this.

ANTHELMINTIC RESISTANCE (AR)

Around the world, AR is very common - particularly in *Haemonchus* and *Teladorsagia*, and to all classes of anthelmintics with the current exception of spiroindoles. As a result, sheep and goat rearing is being threatened in many countries and regions. By the time AR is clinically apparent (i.e. failure of treatment to improve the health of the animals being dewormed), it is well advanced in the flock. Prevention of development of AR is critical for the survival of the small ruminant industries. The following will explain how AR develops and strategies to avoid its development.

DEFINITION OF AR

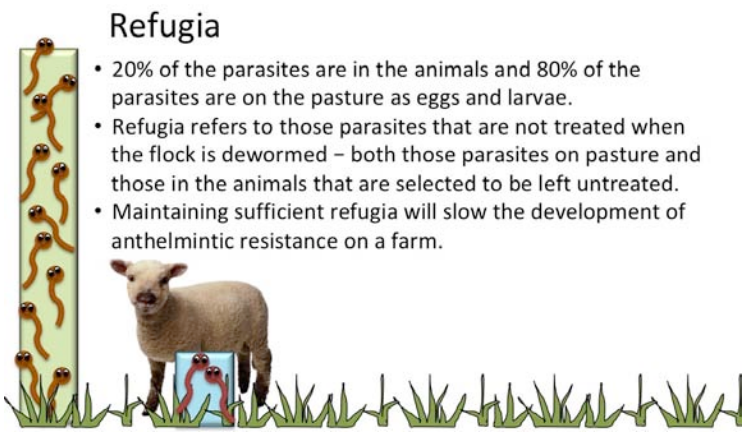
Resistance or AR is the heritable ability of the parasite to survive a normally effective dose of an anthelmintic. Usually a parasite is considered resistant if it survives a normal dose of a single anthelmintic. However, parasites will also survive if the treatment is administered incorrectly - this is not AR but drench failure (see above). Because resistance is a genetic trait in the parasite, the parasite may be homozygous resistant, i.e. having two copies of the genes for resistance (RR), or heterozygous resistant, i.e. having only one copy of the gene for resistance (Rr).

The homozygous resistant parasite is much more resistant to an anthelmintic than the heterozygous parasite. Heterozygous resistant parasites are still susceptible to the correct dosing of an anthelmintic, but will survive if the animal is under-dosed. However, a homozygous resistant parasite may not be affected at all, although repeated dosing at a high level, or dosing with two anthelmintics simultaneously, may be effective for a while. The homozygous resistant parasite is rare in an unselected population of parasites. But once the selection has occurred, parasites do not lose their resistance.

HOW DOES AR DEVELOP ON A FARM?

Refugia explained

The term refugia is applied to the free-living stages of GIN on pasture, i.e. eggs, L1, L2 and L3 stages of larvae as well as the parasitic GIN in the sheep/goats that are not exposed to an anthelmintic treatment. Traditionally a higher proportion of the total parasite load on a farm is on the pasture (80%) as eggs and free-living larvae, compared to the parasite load in the animals (20%), which is comprised of L4, immature and adult parasites. The refugia are the farm's source of susceptible parasites. Elimination or severe reduction of refugia will hasten the development of AR. However, this has to be balanced with the risk of having a high level of pasture contamination, which is a primary cause of sheep/goats developing clinical parasitism. It is important to learn how to ride the fine line between too many parasites in refugia, and too few.



Pressure of anthelmintic use

Repeat dosing with an anthelmintic will often kill 95% or more of all GIN in an animal. But it is the surviving, genetically resistant population that will continue to lay eggs and contaminate the pasture. Repeat, frequent dosing, particularly if under-dosing occurs, will hasten the development of a resistant refugia. While sheep/goats will clinically respond to a drench that is less than 95% effective, eventually the susceptible parasites are in the minority and the drench ceases to be clinically effective. This does not happen overnight, and may take years for a farm to get to this state.

Side resistance

If the parasite is resistant to one drug, then it is resistant to all drugs in that drug class, e.g. if resistant to fenbendazole, then it is also resistant to albendazole. Moxidectin may work for a year after ivermectin stops working, only because it is more powerful than ivermectin rather than a different drug class.

Parasite fitness

It appears that AR can develop more quickly if the population of parasites is already resistant to one or more classes of anthelmintics. It may be that those parasites have the ability to more quickly metabolize drugs than those that are 100% susceptible. This may play a role in the development of multi-class resistance on a given farm. In an Ontario study conducted in the summers of 2010 and 2011, we saw multiple drug class resistance more often than single drug resistance, even on farms that have not used one of those drugs for years.

Consequences of having low levels of refugia on the farm in the face of an aggressive deworming program

Having susceptible refugia on pasture allows the flock the opportunity to become infected again with susceptible parasites - thus lowering the risk of AR becoming a farm problem. However, it is also important to make sure that pastures are not heavily contaminated, so our parasite control practices include lowering the level of parasites in refugia (see later in the handbook for those methods). However, if an aggressive deworming program is also instituted in the face of low refugia, the development of AR is accelerated. Two examples of particularly risky methods of parasite control are:

"Dose and Move" pasture rotation

The "dose and move" strategy was historically designed to prevent animals from carrying parasites into a safe pasture, or one with very low levels of parasite larvae and eggs. But the thinking has changed on this. See Figure 3.

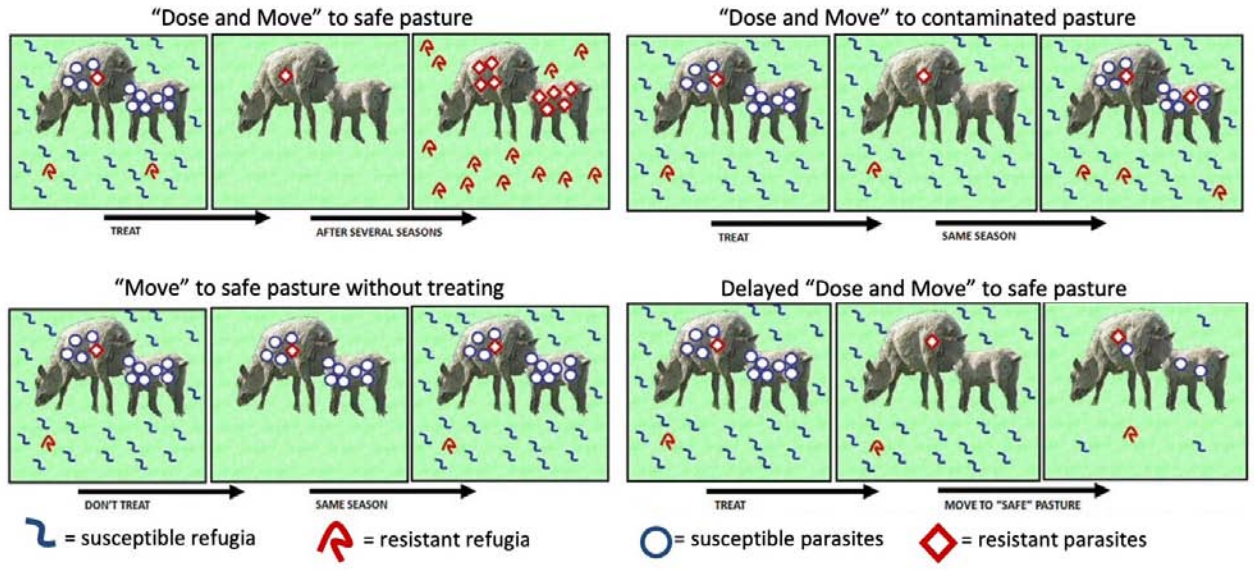
"Dose and Move" to a safe pasture. Deworming sheep and goats on a contaminated pasture and then moving them to a safe pasture will allow animals to maintain a low infection status for longer. However, the parasites that have survived treatment in the animals are resistant to the deworming product that was used. These surviving parasites will then contaminate the pasture with resistant L3. The build-up of resistant parasites to a level where disease and treatment failure is evident, may take several grazing seasons – but when this happens, it is often too late to reverse. For this reason, it is strongly recommended <u>not</u> to move the animals right away.

"Dose and Move" to contaminated pasture. If the animals are dewormed and either left on the same contaminated pasture or moved to another contaminated pasture, the development of AR is slowed considerably as there are plenty of susceptible parasites in refugia. However the animals quickly become infected and must be dewormed more frequently. Eventually, resistant parasites will build-up on pasture over several grazing seasons, and AR will develop, just more slowly.

"Move" to a safe pasture without treating. Sheep / goats are not dewormed but simply moved to a safe pasture instead. For the short-term, they will not pick up more parasites and so the level of infection won't reach the stage where the parasites cause disease. If the farm has many safe pastures, it is possible to rotate the animals through the pastures quickly (e.g. < 2 weeks per pasture) and so keep the level of parasites low. But most farms don't have that many safe pastures and so eventually the animals must return; the pastures become increasingly contaminated and the parasite load in the animals may reach a level that requires deworming to prevent disease.

Delayed "Dose and Move" to safe pasture. This is the preferred approach. To prevent disease in the animals, they are dewormed when infection levels reach high enough levels. But to prevent the development of AR, the sheep / goats are left on the pasture for a <u>few days</u> to pick up susceptible larvae – enough so that they become slightly re-infected with parasites. This dilutes the resistant parasites being shed on pasture while still keeping low levels in the animals.

Figure 3



Dose at lambing / kidding

While *Teladorsagia* and *Trichostrongylus* L3 overwinter well on pasture in our climate, *Haemonchus* does not. This sounds like good news because spring pastures have very low levels to no contamination with this parasite. However, if we deworm all adults prior to turnout (e.g. if we treat all ewes or does at lambing/kidding) we will eliminate all the *Haemonchus* on a farm – with the exception of *Haemonchus* in the animals that have resistance to the anthelmintic used. So this scenario is very similar to "dose and move" to "safe" pasture immediately after deworming. These resistant *Haemonchus* then infect lambs and kids. It may take a very short time for AR resistant *Haemonchus* to become predominant on a farm, even if the adults are rarely dewormed and may occur more quickly if they are dewormed while still in the barn.

Introduction of resistant parasites

Purchase of sheep or goats that contain large numbers of resistant parasites, may introduce AR to a farm - which when combined with improper parasite control measures, will hasten the development of AR on a farm. Goats are a particular risk, as AR tends to develop more quickly with this species. This is because many anthelmintics are metabolized more quickly in goats than sheep (increasing the risk of sub-therapeutic dosing) and because adult goats do not develop immunity as well as sheep do, so they carry more severe infections. Quarantine of new introductions and proper deworming of new introductions is an important strategy to prevent introduction of AR. For details, see ★4 of the 5 STAR WORM PLAN.

Improper treatment

There are many ways to improperly administer a treatment (see above for prevention of treatment failure). By under dosing, heterozygous resistant parasites are more likely to survive, which will increase the number of parasites left in a sheep / goat and hasten the development of AR on a property.

THE CENTRAL CANADIAN SITUATION

Since 2006, a number of studies have examined the epidemiology of gastrointestinal nematode parasites on Ontario farms. Abstracts of these papers are in Appendix 5. This has allowed us to better understand these pathogens in our cold continental climate. Other work – published and yet-to-be published - has been done in Quebec, Nova Scotia and Alberta. This and anecdotal evidence suggests that what we have found in Ontario, is likely also to be found across Canada.

What we found was that anthelmintics are not administered frequently in our flocks but that anthelmintic resistance is very common in *Haemonchus contortus*, to both MLs and benzimidazoles. We speculate that deworming when sheep are in the barn in the winter (e.g. at lambing) is a major factor in this. This means that there are no parasites on pasture for *Haemonchus* on the farm and if all the flock is dewormed, there are no refugia of parasites except those that are resistant. In this manner, anthelmintic resistance can develop very quickly in *Haemonchus*; care must be taken to leave an untreated proportion of the flock and / or to assure sufficient pasture-based refugia to maintain a susceptible population of *Haemonchus*. This will be discussed in more detail in the 5-Star Worm Program.

DETECTING THE PRESENCE OF AR ON A FARM

If AR is suspected in a flock or herd, it is important to review the treatment protocols to make sure that the drug is being administered properly. To confirm AR on a property the following methods can be used:

DRENCH RESPONSE TEST

This can be performed with only 1 faecal sample collection time but only suggests, rather than proves, that AR is present. The group is treated and faecal samples are collected from 10 randomly selected lambs or kids after a period of time (10-14 days for BZ, 14 days for ML, closantel and Startect). Failure to achieve low counts may indicate AR, or treatment failure from other causes.

FECAL EGG COUNT REDUCTION TEST (FECRT)

The FECRT is often used as the "gold standard" for determining if AR is present on a farm. It is scientifically sound when done correctly and will give an accurate picture of how effective anthelmintics are on a given farm. However, the process is labour-intensive and expensive - but is low-tech and gives the best information. If you decide to perform a FECRT, the following protocol is followed:

1) A minimum of 30 lambs/kids or young adults (first grazing season) with elevated faecal egg counts are required, ideally all from the same grazing group. They should not have been treated in the previous 30 days.
2) Individual minimum FEC of 200 epg is required but 300 epg is preferred.
3) Ten to 15 lambs/kids are randomly assigned to each control and treatment group. It is necessary to use this many animals per group because of the normal variation in egg output between animals. It is very important that the assignment be random!
4) If three anthelmintics are being evaluated (e.g. ivermectin representing the macrocyclic lactones (ML), fenbendazole representing the benzimidazoles (BZ) and closantel (CL)), then four groups (60 animals) are needed (e.g. control group (no treatment), ML group, BZ group and CL group).
   a) Keep in mind that closantel will only eliminate *Haemonchus* and so it may be prudent to include either ML or BZ with CL as a combination treatment. Include closantel if *Haemonchus* is the parasite of concern.
   b) If using as a combination treatment, give the drugs in separate drenching guns. Don't mix in one gun.
5) Individual faecal samples are obtained per rectum on day 0 (treatment day).
6) The lambs/kids are weighed using a scale and treated appropriately by drenching to the exact weight of the lamb / kid.
7) The control animals are not treated.
   a) A control group is not needed if a sample is taken on day 0 from all animals in the treatment groups, but if there are lots of animals, it is helpful to include an untreated (control) group.
   b) If a control group is used, then there is no need to sample the separate treatment groups prior to treatment. However a pre-assessment FEC using a pooled sample representative of all the treatment groups, should be done to make sure the level of infection is high enough to perform the FECRT, i.e. a minimum of 200 epg for the pooled sample.
8) All of the animals are returned as one group, to the same pasture to graze.
9) All the animals are sampled again later (10-14 days for BZ, 14 days for ML and CL).
10) The post-treatment faecal egg counts are statistically compared to the control. This is usually done on pooled results rather than individual.
    a) The formula that has been shown to be most accurate is to firstly change any 0 epg results to 25 (if using the McMaster method – this is called a bias correction factor) and then do an arithmetic comparison of the means.
    b) Because the statistical analysis can be complicated, web-based software can be used by uploading the data on a spreadsheet. http://shiny.math.uzh.ch/user/furrer/shinyas/shiny-eggCounts/
11) Failure to reduce by 95% or greater compared to the control indicates resistance when this program is followed.
    a) Confidence intervals (CI) are also calculated and AR is present if the lower CI is < 90%.

Other research methods for determining presence of AR

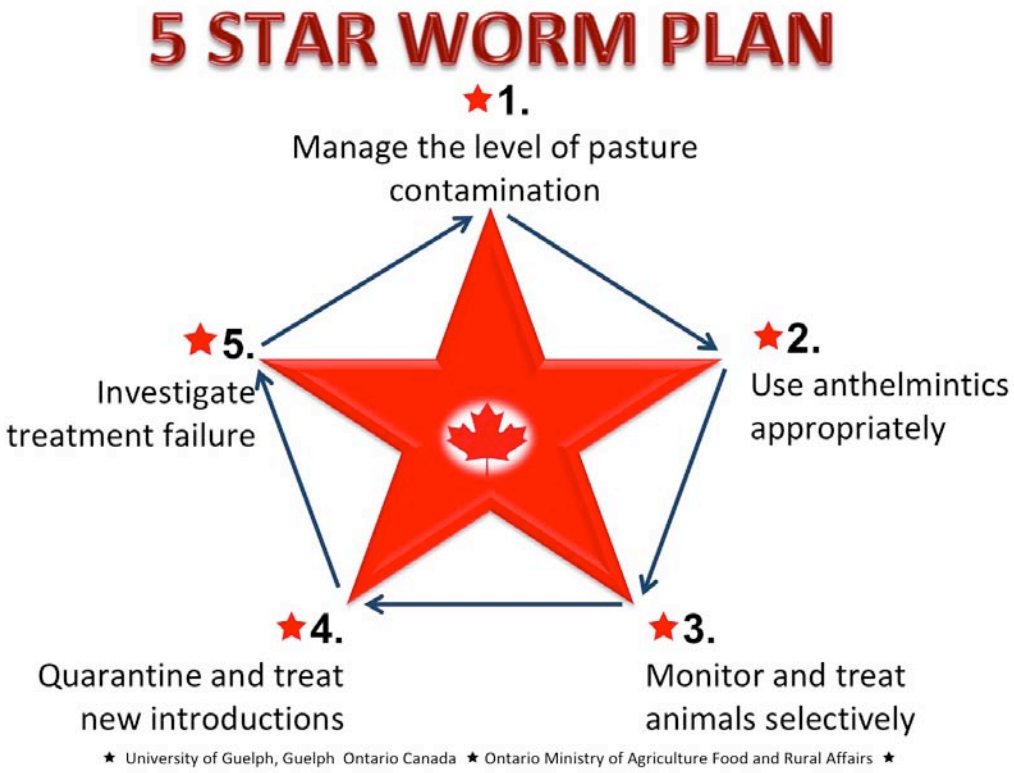
Larval development assays (LDAs) can be used to detect AR in the laboratory but have some drawbacks. They cannot be reliably used to detect ML resistance, are expensive, and require a specialized laboratory to work properly. Eggs are hatched and developed to the L3 stage while exposed to an anthelmintic. The level of successful hatching, development or feeding is then measured, depending on the test used. The positive aspects of the assays are that they require less labour on farm and don't require treatment or handling of animals. At this point, LDAs are mostly used as a research tool.

Exciting work has been done on identifying the genes in parasites that code for AR to the different anthelmintics. A PCR could then be used on L1 (hatched eggs) to detect these genes and determine the prevalence within a parasite population. This work is on-going at both McGill University and the University of Calgary.

THE 5 STAR WORM PROGRAM

The goal of a parasite control program is to control the level of parasites on the farm to a level that has minimal effect on animal health and productivity, without allowing the development of anthelmintic resistance.

This is known as sustainable Integrated Parasite Management or sIPM. Gastrointestinal parasites do not need to be an issue on a small ruminant farm, if sound principles and an understanding of the epidemiology are used in developing a suitable control program. We present the main points here. The program is called "5 STAR WORM PLAN". Use this program with your veterinarian to develop a health management approach that is correct for your farm.



★ 1. MANAGE THE LEVEL OF PASTURE CONTAMINATION

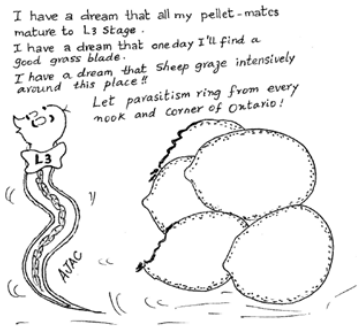
There are many methods available to reduce the level of parasite contamination of pastures. None in of themselves are 100% effective, but together they are very important in any sustainable integrated parasite management program and will make the difference between success and failure. The goal is to have all pastures as "safe" pastures; there are many methods to achieve this.

1.1 MANAGE THE BIGGEST SOURCES OF PASTURE CONTAMINATION

The two biggest sources and times of pasture contamination with GIN eggs are: 1) lambs and kids by mid to late grazing season (e.g. late July / August) and 2) adult females in late gestation and lactation (the periparturient egg rise) – usually in the spring at turnout. To manage the contamination by youngstock, monitor frequently and treat when needed– particularly from mid-grazing season (e.g. early to mid-July). To manage the contamination from the PPER, treat adults selectively before putting to pasture (see below). Managing parasitism in farms which practice pasture-based lambing / kidding is more difficult because of the risk of mismothering if ewes / does need to be gathered up to treat at or soon after giving birth. In that case, treatment may need to be delayed but it is important to include a method of monitoring the adults and treating when needed. Young, nursing lambs and kids don't tend to pick up parasites in the first few weeks of life because they are not grazing as much, but the parasites deposited by the adults will wait for them.

1.2 USE OUR UNDERSTANDING OF THE BEHAVIOUR OF THE FREE-LIVING STAGES

By understanding where the infective L3 are in the pasture, we can modify grazing management to try to reduce exposure of the sheep / goats to them.



1.2.1 Break up the faecal pellet

Remember that L1 and L2 stages live inside the faecal pellet. If the pellet is exposed to moisture or is broken up by earthworms, dung beetles or by mechanical means (e.g. harrowing), the L1 & L2 are then exposed to the environment where adverse temperatures, sunlight or dry conditions can kill them more rapidly. Unfortunately, this also facilitates release of L3 from the pellets.

1.2.2 Modify grazing based on temperature and humidity

L3 dislike dryness so they will migrate down to the soil during hot days but migrate up when the dew is on the grass. Waiting until the dew is off the grass to graze short pastures is often not practical. Exposing the ground to sunlight may have some benefits as it gives the L3 less place to hide and exposes them to heat and drying. This can be done by either de-thatching using a harrow, or routinely planting new pastures and ploughing in old pastures.

1.2.3 Modify grazing based on sward height

The L3 are restricted on how high they can climb (usually not higher than 5 cm or 2 inches), so long grass pastures may be safer than short grass. However, sheep and goats are able to more selectively graze than cattle and will avoid long grasses with higher lignin content in favour of the new shoots close to the ground. Overgrazing pastures will increase the infection rate by forcing the livestock to graze close to the soil.

1.2.4 Eliminate areas of the pasture that favour L3 survival

Areas of the pasture that are wetter, such as low lying areas or around water troughs, may have greener grass and attract sheep and goats to graze but also favour survival of L3. If practical, eliminate these areas for grazing either by fencing off or gravelling (e.g. around water troughs). If there are bedded areas on pasture (e.g. around round bales), these will become heavily contaminated.

1.3 ROTATE PASTURES WITH OTHER LIVESTOCK SPECIES

While cattle share some parasites with sheep and goats (e.g. *Trichostrongylus axei*), rotation with this species has been shown to lower pasture infectivity to sheep and goats. Horses will also work but not llamas as they share parasites. Co-grazing (grazing at the same time) with cattle is less effective but may help.

As with all methods of lowering pasture contamination, if deworming is done so there are few parasites in refugia, this practice will increase the risk of development with AR.

1.4 AVOID GRAZING SHEEP AND GOATS TOGETHER

Sheep and goats share the same parasites. Because adult goats do not develop immunity to parasites they will be a more important source of pasture contamination than adult sheep. Additionally, they metabolize anthelmintics more rapidly than sheep, require higher dosages than sheep and because of that are at risk of developing AR more rapidly than sheep.

1.5 REST PASTURES THAT ARE HEAVILY CONTAMINATED

If a pasture was particularly heavily infected at the end of the previous grazing season, select it for ploughing, reseeding, haying and / or grazing with another species.

1.6 BE AWARE OF RISKS OF CONTAMINATION FROM STORED MANURE

Although it is unlikely that infective larvae will survive in well-composted manure that has heated properly, fresh manure can be a source of parasites. A particular risk may occur in the spring when bedding packs are cleaned from the barn and spread directly to fields intended for grazing or hay production. Thus, it is safer to spread manure onto fields prior to ploughing for crops.

Often manure is seasonally stored on a cement or gravel yard. Make sure that the animals have no access to this pile and additionally make sure that runoff from the manure pile doesn't enter any place where livestock are maintained. This includes yards and pastures. This should be included in any farm's nutrient management plan.

1.7 USE LOW-RISK PASTURES FOR THE MOST SUSCEPTIBLE ANIMALS

Graze weaned lambs/kids on newly seeded / rested pasture or hay fields. Annual pastures (e.g. turnips) that are ploughed in at the end of the season are also low risk.

1.8 "DOSE AND MOVE" VERSUS DELAYED "DOSE AND MOVE"

If the entire flock is to be treated, there are techniques explained in Figures 3 that allow a susceptible refugia to be maintained – ideally, dose and delay moving for a few days is preferred to ensure animals become infected with a low dose of susceptible parasites.

1.9 REDUCE THE CONTAMINATION OF A PASTURE BY USING PASTURE ROTATION

Firstly, understand that L3 can survive for weeks to months on pasture if the environment is moist and temperate, and that the L3 of most species (except *Haemonchus contortus* that does not survive < 0° C.) can survive over winter. This makes it very difficult to accurately predict when a pasture is finally "safe" in a pasture rotation system. Most pasture rotation systems require that the flock return repeatedly to the same pasture in a grazing season. Unless the frequency of rotation is < 7 days (*Haemonchus*) or < 14 days (*Teladorsagia* and *Trichostrongylus*), eggs deposited when grazed previously will likely be hatched and developed to L3 – just waiting to infect the returning livestock. Additionally, those L3, under the right weather conditions (e.g. temperate and moist), will survive for months. The following are some suggestions that will help with using pasture rotation to control parasites:

1.9.1 Evasive Grazing

This technique of grazing pastures when the risk of parasites is lowest requires knowledge regarding the speed of larval development given local conditions. Models have been developed that take geography, weather and management practices into account to predict when pastures might be becoming dangerous. These are used in countries such as Australia, New Zealand and the UK (http://www.nadis.org.uk/parasite-forecast/). It should be remembered that during the summer, pastures can remain very infective for months, making evasive grazing impractical if used as the sole method for parasite control.

1.9.2 Intensive Rotational Grazing

This is a form of evasive grazing and is relatively safe if the following hold true:

1. Animals are moved from the strip before eggs hatch and the larvae develop to the L3 stage (variable depending on weather; longer in cool weather but shorter in warm weather); AND

2. Animals do not return to the strip until the L3 have died (variable depending on weather and moisture but may be up to several months if temperate and humid). Most producers cannot afford to do this.

On average, the worst time period between grazing sessions is 2 to 3 weeks - the most likely time that the eggs have hatched and developed to L3. While short-term grazing will limit pasture contamination, returning to the strip several times in a season will result in the build-up of L3 - just as if the pasture was set stocked (i.e. animals put to the same pasture for the entire grazing season). While it may be prudent to intensively rotationally graze in order to make optimal use of the pasture, monitor FEC closely.

1.10 IF HEAVILY CONTAMINATED PASTURES MUST BE GRAZED

Often, the producer has only heavily contaminated pastures available for grazing. The following strategies may help to lower risk in the face of grazing heavily contaminated pastures.

1.10.1 Rotate weaned youngstock onto safe pastures ahead of adults

After weaning, lambs or kids should have "first access" to any safe pastures on the farm. This way there is less risk from exposure to contaminated pastures. Adults are better able to tolerate heavily contaminated pastures.

1.10.2 Use adults to graze heavily contaminated pastures

If pastures are heavily contaminated and safe pastures are in short-supply, non-lactating ewes or does not in late pregnancy can be grazed more safely than youngstock on these pastures. If the adults have good immunity, this may help to lower the infectivity by grazing off L3 but not recontaminating the pasture with fresh eggs. This should be done while monitoring FEC.

1.10.3 Don't graze nursing lambs / kids

It is more difficult to manage the parasite exposure of lambs / kids when grazing with their dams. If only heavily contaminated pastures are available, try to avoid grazing nursing lambs/kids. If possible, practice early weaning (e.g. 60 days) so their exposure can be better managed. If not, increase the frequency of FEC monitoring. At weaning, lambs / kids should be moved to pasture with the lowest level of contamination.

1.10.4 Lower stocking densities

Because of the nature of faecal consistency in small ruminants (pellets as opposed to soft patties), sheep and goats often do not have the option of grazing away from faeces, as cattle do so controlling stocking density becomes more important with these species. By lowering stocking densities, there will be less pasture contamination with feces. Recommendations vary but keeping set stocking densities < 6 to 8 sheep/goats per acre is often mentioned. With rapid pasture rotation, these densities can be increased. But FEC monitoring must also be done regardless of stocking density.

1.11 RECORD PASTURE USE AND TREATMENTS.

Appendix 3 is an example form that can be used for this purpose

★ 2. USE ANTHELMINTICS APPROPRIATELY

2.1 TREAT APPROPRIATELY TO AVOID TREATMENT FAILURE AND DEVELOPMENT OF AR

- Weigh the sheep/goats. Dose for the heaviest in the group.
- Use only dewormers with a Drug Identification Number (DIN) to assure quality of the product.
- Dose correctly by reading the label and calculating based on the weight of the animals to be treated.
  - If not labelled for sheep or if using in goats, obtain the correct dosage from your vet.
  - Goats are generally treated at 2X sheep dose (BZ) or 1.5X sheep dose (ML).
- To increase effectiveness of a drug when AR is suspected:

- Do not double the recommended dosage but rather give the recommended dosage twice 12 hr. apart (BZ and ML only).
- Holding the sheep/goats off-feed for 12 to 24 hr. before treatment with a BZ can increase the length of time that the anthelmintic is effective.

- Calibrate the drench gun or automatic syringe frequently.
- Drench correctly by depositing the entire dose over the tongue, at the back of the throat.
- Oral drenches should be used instead of injectable products.
- Use the correct route of administration for the product. Do not use a cattle pour-on.

2.2 Rotate anthelmintic classes slowly

Consensus suggests to not rotate anthelmintic drug classes more frequently than annually. Rapid rotation is thought to lead to multiple class AR. Do not use an anthelmintic until it doesn't work anymore due to anthelmintic resistance. This will mean the loss of that drug class permanently for that flock.

2.3 Using combination dewormers

If resistance is present for a drug class on a farm, sometimes combining it with another drug class will increase its efficacy – at least for a while. In Canada, a combination dewormer is licensed for use in sheep (Startect, Zoetis Canada), the only one licensed in North America. This dewormer can be used for quarantine drenching to prevent introduction of resistant parasites (see ★4). It can also be used judiciously when resistance to BZ or ML is present on a farm or to lower the risk of AR developing. It can't be used on goats, however.

There is evidence that when using a novel-to-the farm anthelmintic in combination with another class, the development of AR is very delayed. If the decision is made to use two anthelmintic classes at the same time (with the exception of Startect), do not combine in the same drench gun but give separately in sequence at a full dose for each product, i.e. drench the sheep with drug class A (e.g. ivermectin) and using a different drench gun, drench with drug class B (e.g. fenbendazole). Do this only on the advice of your flock veterinarian, including calculation of a proper meat withdrawal period since combining deworming treatment constitutes extra-label drug use (ELDU) and may extend meat withdrawal times. Do this while also re-evaluating your farm's parasite control program so that further development of AR is avoided.

★ 3. MONITOR AND TREAT ANIMALS SELECTIVELY

3.1 TARGETED TREATMENTS (TT)

This means to treat sheep or goats **only when the group needs it**. This is done by monitoring FEC (usually pooled samples) and clinical evidence of disease and then treating the group. Record all FEC results (example form provided in Appendix 4). Increasing the interval between anthelmintic treatments reduces the development of AR. Times to monitor (and possibly treat):

3.1.1 Ewes / does prior to lambing / kidding

This is to eliminate or reduce the PPER, which is considered one of the most important early sources of pasture contamination for lambs and kids. Monitor ewes and does at lambing or early lactation. Ewes / does with a significant PPER will contaminate the spring pasture with eggs starting a few weeks prior to lambing / kidding and continuing through to about 6-8 weeks into lactation depending on nutritional management. Youngstock will be very susceptible to infection from any overwintered L3 on pasture.

Ewes may suffer disease or decreased productivity if not dewormed in the face of a high FEC. Does may also be more prone to disease as immunity in this species is poor for parasites. However, treatment of the entire group or when treatment is not needed may hasten development of AR on a farm – particularly for *Haemonchus*. This is because there are not sufficient parasites in refugia (in the case of *Haemonchus*, no L3 refugia on pasture in the spring) to prevent development of AR. Under some management conditions, e.g. if ewes lamb and nurse lambs indoors, it may not be necessary to deworm at this time because they are not contaminating pasture – as long as the females appear healthy.

For these reasons, it is important to make the decision on whether or not to deworm prior to lambing / kidding with your flock/herd veterinarian and to always monitor faecal egg counts in nursing adults.

3.1.2 Lambs/ kids at mid-summer

The exact date to start taking faecal samples will vary depending on the previous parasite history of that farm, the warmth and humidity of the summer and how warm weather arrives on that farm. Generally, early to mid-July is the earliest that we routinely see clinical evidence of parasitism. Mostly it is slightly later - late July to August, which appears to be the highest risk period in our climate for haemonchosis. However, there is tremendous variation in the start of warm weather in Canada and veterinarians and producers should be ready to make adjustments to these dates.

GI parasitism can be controlled if animal infection is routinely monitored. This is done by checking FECs in the youngstock (and adults if grazing together) in early to mid-July, and treating only when high counts are found (or in the case of haemonchosis, evidence of anaemia can also be used, i.e. FAMACHA© scoring). If the FEC is negative, but animals are showing severe clinical signs of parasitism, consult your flock/herd veterinarian to determine if another disease is present (e.g. coccidiosis). Alternatively, the prepatent period for the parasite may not have been reached yet.

Occasionally, if the spring pasture is heavily contaminated from the previous grazing season with over-wintered L3 (usually *Teladorsagia* but not *Haemonchus*), parasitism can occur earlier. Lambs / kids on these pastures may encounter such a severe infection by grazing these overwintered L3 that they become clinically ill (diarrhea, off-feed, depressed) from the immature parasites before eggs are present in their feces. Any youngstock that die should be necropsied by a veterinarian to determine if this is the cause of death. Never assume death is due to worms (or that it isn't!).

3.1.3 Repeat monitoring in the grazing season

Monitor frequently at the times of highest risk, i.e. mid-summer to early fall. If when the lambs/kids are monitored, the FEC is below the cut-point to treat, resample in mid-summer at least every 3-4 weeks and more frequently, particularly if *Haemonchus* is known to be a problem in the past or the FEC is borderline. On farms with a history with *Haemonchus*, it may be necessary to monitor as frequently as every 10 days.

3.1.4 Monitor after treatment

Routine faecal egg counts should be done every 4 to 6 weeks after treatment (shorter periods if a less persistent anthelmintic is used) although this can be done more frequently if pastures have significant contamination and no safe pastures exist on a farm. If the animals with signs of parasitism do not rapidly improve after treatment, it is strongly recommended to resample at 14 days to determine if treatment failure occurred (see above). Keep in mind there are several reasons for treatment failure beyond anthelmintic resistance so also review all your protocols for administering a dewormer.

3.1.5 Monitor according to farm history

By knowing the farm history, the time of monitoring can be adjusted. For example, if the previous summer, lambs had elevated FEC in the first week of July, then monitoring should be started in mid-June.

3.1.6 Monitor in the autumn?

By October there is less reason to use FECs to determine infection. Although the animals may be parasitized, most of the development is now to the arrested stage (L4), which do not feed or produce eggs. Although there is variation, adult parasites naturally die off in the autumn with only a few surviving into the winter and those do not produce many eggs. Performing FECs at this time will not properly estimate the level of infection present in the animal. However, if there is concern because of clinical evidence of disease or there were problems in previous years, a FEC can be done; a high count is significant but a low count doesn't mean the animals are not parasitized, just not with egg-producing adults. Similarly, FAMACHA© score can be used to augment FEC to determine level of *Haemonchus* infection.

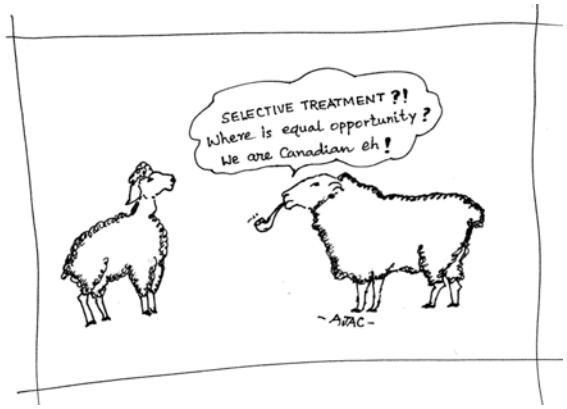
3.1.7 Treatment of breeding animals at housing?

Treatment in the autumn may reduce the arrested L4 that overwinter in the animal, and thus may lower the level of PPER the next spring in pregnant ewes and does. But we need to be assured that the treatment is both necessary and actually works at this time of year. Treating the entire flock may lead to AR in *Haemonchus* because of lack of parasites in refugia. For this reason, it is strongly recommended to only selectively treat animals at this time.

3.1.8 Treatment pre-breeding?

The recommendation to treat pre-breeding should only be done if monitoring or poor condition suggests that the adults are parasitized. Usually adults will not show signs of parasitism unless periparturient or debilitated with another disease or poor nutrition. Treatment when not necessary will contribute to the development of AR.

3.2 TARGETED SELECTIVE TREATMENTS (TST)



This means treating **only those individual animals that need it when they need it** and is based on the knowledge that in any given population, only a proportion actually requires deworming. The challenge is to correctly identify the animals that need treating and those that don't. The development of AR can be slowed or prevented if about 1/3 (30%) of animals are NOT treated. This proportion can be lower if no AR is present but if AR is already present, then 30% is the minimum proportion that should be untreated. This approach leaves a susceptible parasite population in refugia – both on the pasture and in the untreated animals and is critical to the success of any sustainable integrated parasite control program. The producer has only a few options to be able to do this effectively and economically.

3.2.1 Using faecal egg counts

Unless the flock size is very small, it is not economical to perform individual FEC on all animals in order to detect the "big shedders", i.e. those 30% of animals that shed 70% of the eggs. There is no method of determining parasite egg load in faeces other than using a laboratory based-test. Some producers wish to perform their own FECs. If this is the case, they must be well trained and focus their labour on performing FECs when the most information can be gained.

3.2.2 Using the FAMACHA© system

The FAMACHA© system can be used very effectively to select individual animals for treatment of haemonchosis - but is not effective at detecting infection of other GIN species. It could be used on farms that know when *Haemonchus* becomes a problem (e.g. starting late July, early August) but **must** be combined with pooled FEC to rule out other causes of parasitism. Producers should be properly trained (see above) before undertaking this procedure. Fatal errors (not treating an animal that is anaemic because it was scored incorrectly) can be reduced with proper training.

Sheep or goats that score 4 or 5 require treatment (score 3 as well if a large part of the flock is anaemic) and then everybody monitored every 2 to 3 weeks during the high-risk period. FAMACHA© cards must be used in good light, and replaced annually as the colour may fade with time. Because there are other causes of anaemia, it is important to investigate treatment failure. Use the provided record to track FAMACHA© treatment results.

3.2.3 Using evidence of diarrhea

Dag scores indicating diarrhea may be helpful when the producer can eliminate other reasons for scouring (e.g. coccidiosis or lush pasture) and may work best when combined with monitoring weight gains. However some research suggests that by the time the lambs or kids have diarrhea, significant clinical disease is occurring - i.e. waiting until they have diarrhea is too late. Additionally, haemonchosis can be severe without signs of diarrhea.

3.2.4 Using weight gains

Routine weighing of lambs or kids (e.g. every 3 weeks) can identify those animals that are not gaining as fast as their cohorts, one reason for which may be GIN parasitism. One method of using this information is to only deworm the lighter animals and leave the heavier ones untreated. Expected ADGs will vary depending on the breed, sex and age of the animal and the pasture being grazed. A producer may get a feel for what growth should be expected from the youngstock on a particular type and growth of pasture. That may be more useful than a scientific formula.

For those using RFID and automated weighing systems, it is possible for producers to select poor gaining animals for treatment on a relatively frequent basis. The same type of system is used frequently for lambs in feedlots. That way, changes in the individual animal's weight can be tracked and individuals that fail to gain would be treated. Pooled FEC should also be done to verify that parasitism is the cause of the poor growth and not poor nutrition or other disease such as coccidiosis or pneumonia.

Body condition score was not found to be helpful of predicting FECs in a recent Canadian study. It may be that it is not sensitive enough to pick up early parasitism. By the time the animals are thin, parasites have taken a severe toll. That being said, very thin animals should be treated but also monitored for response to treatment as many other diseases can be responsible for animals with a BCS of < 2, e.g. Johne's disease (paratuberculosis), maedi visna, chronic pneumonia, dental disease and internal abscesses plus many more.

3.2.5 Using milk production in grazing dairy small ruminants

Dairy goats in their first lactation may benefit from deworming in terms of improved milk production if they have a history of grazing as youngstock. While there is evidence that deworming will improve milk production in dairy ewes and dairy does, keep in mind that no anthelmintic is approved for use in lactating dairy small ruminants. If deworming is done, it should be an evidence-based decision, e.g. elevated FEC. The producer and veterinarian are responsible for ensuring that chemical residues are not present in milk sold for human consumption.

Use of an anthelmintic approved in lactating dairy cows in lactating dairy sheep or goats, is not a guarantee that violable residues will not be detected in the milk. This is for several reasons: dose is often higher in goats; anthelmintics are metabolized differently in sheep and in goats than in cows and so excretion may be longer; there is no maximum residue limit (MRL) established for drugs not licensed for lactating dairy sheep / goats so if the test can detect it – even at a level lower than is acceptable for cows, it is in violation. As previously mentioned, have the veterinarian submit a request to CgFARAD to obtain proper guidance.

3.2.6 Using number of lambs / kids nursing

There is evidence that ewes or does nursing multiples shed more eggs than ewes or does nursing singles. This is likely due to differences in nutritional stresses between the two groups. Deworming only those females with multiples - either before parturition based on pregnancy scanning, or after based on number nursing - is one way to target those animals that likely have the highest PPER.

3.2.7 Using FAMACHA© scoring at lambing / early lactation

Research performed in Ontario flocks found that the criteria for selection of treatment (*Haemonchus* specific) in the period around lambing and early lactation, should be FAMACHA© score 3, 4 or 5. If a thin ewe has a normal FAMACHA© score, then it may also be advisable to treat her. It is critical to also perform pooled FECs to make sure that other parasites are not also an issue.

3.3 USING THE "5 POINT CHECK" CRITERIA FOR TREATMENT

This system identifies sheep and goats that may require deworming and was developed in South Africa. It includes infection from a variety of parasites – not just GIN and embraces the concept of targeted selective treatment or "*Leave the best and treat the rest*".

1. The nose is checked for discharge that indicates nasal bots (*Oestrus ovis*)
2. The eyes are checked for anaemia, indicating blood-sucking worms
3. The jaw is checked for submandibular oedema that also accompanies anaemia and protein-losing infections cause by parasites such as *Haemonchus* and liver fluke (see below)
4. The back is checked for body condition score indicating possible infection by internal parasites like *Teladorsagia* and *Trichostrongylus* species.
5. The tail is checked for signs of diarrhea, indicating mainly worms that also cause loss in body condition score.

This approach is still being refined and requires FEC monitoring. Remember that by the time an animal shows signs of parasitism such as in points 2, 3, 4 and 5 – it is already very ill. This system requires that you also work hard to reduce exposure to parasites on pasture (under ★ 1 of the 5 STAR WORM PLAN).

3.4 ALTERNATIVE METHODS OF CONTROL TO REDUCE RELIANCE ON CHEMICAL ANTHELMINTICS

To reduce the use of chemical anthelmintics, some of the following methods have been used to augment targeted selective treatment. Regardless of what methods are employed, make sure they are science-based and can work on your farm. A recent analysis of the published literature in this area found that genetic selection and some nutritional methods were the only scientifically proven means to alternatively control GIN parasitism. The abstract of the publication is presented in Appendix 5.

3.4.1 Genetic selection

The breeding of resistant sheep or goats can be done by selecting a breed (e.g. some tropical hair breeds) or selecting individuals within a breed - usually rams that have lower FEC or other measures when compared to other rams in the group. However, it is important to make sure if "resistant" breeds are selected that one does not sacrifice important economic traits such as prolificacy, milk production, growth and carcass characteristics.

Gene marker tests in some countries will help identify sheep that will have lower FEC although are breed dependent. There is much research ongoing in the use of genomics to identify these animals more accurately. Selecting animals based on their ability to mount an immune response is promising. The CarLA® saliva test (Carbohydrate Larval Antigen) developed in New Zealand (AgResearch) measures antibodies to the L3 stage of GIN and can help to select sheep that develop immunity more rapidly or to cull animals that do not. Recently completed research at the University of Guelph is evaluating its utility under Ontario conditions. From this work (thesis in preparation) it appears that the CarLA® saliva test performed in youngstock in October, can predict which animals have lower FECs and higher CarLA antibodies the following grazing season when those ewe lambs give birth and raise their own lambs. This is very promising and requires more research to determine how heritable this trait is in Ontario breeds and grazing conditions.

Remember that immunity is acquired and resistant animals still need to be infected with parasites to develop this immunity. Heritability for this trait is moderate (h^2 is 0.25 to 0.3), so a producer could use FEC in ram lambs or buck kids (comparison within a group) as a criteria for selecting a replacement male, and marketing those that have high FECs. But because heritability is only moderate, genetic progress within a flock may take up to 5 years to see an impact on flock levels of parasitism. To properly select parasite-resistant males, it is important to have a large enough group to accurately find the resistant animals without sacrificing important genetic traits of production.

Remember that goats in general do not develop immunity as well as sheep. Much less research has been done with goats on selecting for genetic resistance and so the following strategy covered in the next paragraph may work better for this species.

Culling the "worm magnets"

Rather than selecting resistant animals, it is easier to identify and cull parasitized adults that are slow to develop immunity to parasites. This can be done by culling those with a high FEC, repeatedly have scores 4 or 5 on the FAMACHA© chart or require repeated deworming treatments, AKA "worm magnets". These adults should be removed from the breeding flock and lambs/kids from these animals should be sent to market rather than retained as replacements. Additionally, lambs or kids that need repeated treatments should not be retained as replacements as they are also more likely to give birth to offspring with less ability to develop immunity to parasites.

Resilient animals

Resilient sheep/goats will be infected and will contaminate a pasture with eggs, but will not have as significant production losses. There is differing opinion about whether these animals should be kept. Regardless, within a

population, there will be resilient and susceptible sheep so that selection must be done by using good records, measures of anaemia, FEC and growth monitoring in order to avoid losses and to select the correct animals.

It is important to remember that using clinical parameters alone, will not allow identification and treatment of these high egg producing resilient animals. This is another reason why it is important to use FEC to monitor infection.

3.4.2 Pasture plants containing condensed tannins

Grazing pastures containing a large proportion of plants with high levels of condensed tannins (CT), has variably been shown to reduce shedding of eggs in the feces. In North America, most research has been published on the legume *Sericae lespedeza* (SL), a warm climate plant. The mechanism appears to be 2-fold. While there is a direct effect by CT on the ability of the adult parasite to produce eggs and for those eggs to develop to infective larvae in the feces, at least some of the effect is from the elevated levels of by-pass protein available to the animal. Animals fed SL also have an improved immune response over animals on a control diet.

Low levels of CT in the diet have been shown to increase reproductive performance and wool growth independent of parasite load. However, CT can be toxic to the animal if too high; high levels in the diet decrease feed consumption and have a negative effect on performance.

Two temperate climate plants with some potential benefits are Bird's Foot trefoil (*Lotus corniculatus*) and Sulla (*Hedysarum coronarium*). Experimentally, sainfoin (*Onobrychis coronarium*) has been reported as both beneficial and of no benefit. There are other CT plants and tree extracts (Quebracho extract for example) that are promising and may be a helpful adjunct to other control measures. Research on varieties of Bird's Foot trefoil are beginning at the University of Guelph to determine tannin content at different times of the year and at different cutting stages. The next step will be to evaluate palatability and treatment effect in both cattle and sheep.

3.4.3 Nematophagus fungi

A fungus, *Duddingtonia flagrans*, grows in faeces - sending out hyphae that trap and kill the free-living forms of GIN in faecal pellets. While these fungi occur naturally, in order to get them into the feces in sufficient quantity to be effective, the spores must be fed to the sheep daily for a minimum of 60 days. The intent is to feed at turn-out for a period of time to disrupt the build-up of L3 on pasture until the season is advanced enough that disease will not occur in that grazing season. Two products containing *D flagrans* spores are available commercially in Australia and USA (BioWorma® and Livamol® with BioWorma®). More information is available on the company website.

3.4.4 Copper oxide

Copper oxide wire particles were first developed to supplement sheep and goats in areas of the world with copper deficiency. It appears effective in temporarily reducing infections due to *Haemonchus contortus*. It does not appear to improve weight gains (over controls). It does elevate liver copper levels in sheep. Given the copper status of sheep in central Canada where copper toxicosis is already an issue, use of copper oxide wire particles is not advised without monitoring of copper status of the flock. It is also critical to realize that copper sulphate (bluestone) should NEVER EVER be fed to sheep or goats. There have been several cases in Canada of copper toxicity in both sheep and goats related to producers feeding copper sulphate in the mistaken belief it will control parasites.

3.4.5 Vaccination

A commercial vaccine is available in Australia, New Zealand and the UK to control *Haemonchus contortus* (Barbervax). The Moredun Research Institute, a UK-based animal health charity owned by farmers, developed Barbervax in partnership with the Department of Agriculture and Food, Western Australia, with support from Meat and Livestock Australia. All profits are invested in further research projects aimed at reducing livestock disease e.g. a vaccine for scour worms. It is not yet licensed in North America.

The vaccine is killed and is made from whole *Haemonchus* parasites harvested from purposely infected lambs at slaughter. By stimulating immunity, the egg count and number of parasites is reduced, thus lowering pasture contamination. The vaccine requires three priming doses to be administered before the height of *Haemonchus* season. This is followed by two or three additional vaccinations during the grazing season for a total of five to six vaccine doses. Please keep in mind that the research was done in Australia where the grazing season is year round. While 5-6 vaccinations sounds like a lot, in that country it is common that each lamb is drenched with a dewormer 5-6 X per year. Use of this vaccine will

reduce greatly the amount of chemical dewormers needed by reducing pasture contamination and enhancing immunity. Research is ongoing to develop vaccines against the "scour worm" parasites, i.e. *Teladorsagia* and *Trichostrongylus*.

3.4.6 Alternative Dewormers

There have been many alternative or natural deworming products recommended over the years. Some are toxic to sheep and goats as well as the parasites (e.g. nicotine). Some do not work in controlled, peer-reviewed studies (garlic, papaya seeds, diatomaceous earth). Diatomaceous earth may be useful for control of external parasites but more research needs to be done to show sufficient efficacy and safety. It is dangerous for humans to inhale. There are other herbal plants that have been hypothesized to be effective parasiticides, (e.g. Neem oil) but at this time there is insufficient supportive scientific evidence for this claim, and safety for both animals and humans has not been demonstrated.

The purpose of "natural" deworming products is to reduce the use of chemical anthelmintics. While this is a laudable goal, these products – in the absence of proof of efficacy (or safety) – may pose a threat to the welfare of the animals. The goal of the 5 STAR program is to reduce the use of anthelmintics and should be implemented with the objective of greatly reducing chemical use but not allowing for animals to suffer from the effects of parasitism.

★ 4. QUARANTINE AND TREAT NEW INTRODUCTIONS

Purchased sheep or goats may introduce parasites, and possibly AR. While performing a FEC may determine if infection is present, it may be more prudent to effectively treat the animal(s) while in isolation and then expose the animal to the farm parasites prior to mixing. Because these recommendations are very farm specific, you must involve your flock veterinarian in developing this protocol. Below are suggestions as to how this may be done.

4.1 TREAT ALL NEW INTRODUCTIONS WHILE IN ISOLATION

Purchased sheep and goats should not be turned out with your flock or onto pastures grazed by your flock until the possibility of AR parasites has been minimized.

4.1.1 Unknown history of AR in the farm of origin

Startect™ (Zoetis Canada Ltd) is licensed for sheep and should be the treatment of choice for a quarantine treatment in that species. It should not be used in goats, as there is no safety or efficacy data in that species. To treat any animal, they should be weighed first and dosed to body weight or slightly higher to assure there is no risk of under treatment.

For goats, treat with ivermectin drench (not injection) at 1.5 X the sheep dose. After the animal has swallowed the anthelmintic, follow-up this treatment with a BZ anthelmintic (don't mix together) - either fenbendazole or albendazole (not in does that may be in their first 30 days of pregnancy) at 10 mg/kg bw (double the sheep dosage). The BZ treatment can be repeated in 12 hours on the advice of the flock veterinarian.

4.1.2 Resistance to both ML & BZ dewormers is present in the farm of origin – goats only

If the parasite of concern is *Haemonchus*, then closantel (Flukiver™, Elanco Canada Ltd) will likely be very effective, as resistance has not yet been reported to this anthelmintic, although this product is not approved for goats in Canada and should only be used under the guidance of the herd veterinarian. In Canada, the vast majority of AR has been identified in that parasite and not in *Teladorsagia* or *Trichostrongylus*. Use of ivermectin or a BZ (not both) in combination with closantel as described in 4.1.1 will most likely eliminate those parasites.

4.2 HOLD TREATED ANIMALS OFF PASTURE

The sheep / goats should be held off pasture and ideally in a drylot for at least 48 hours to allow passage of any parasite eggs. Manure from this holding time needs to be properly composted so that the resistant eggs and larvae are killed.

4.3 TURN ANIMALS ONTO A CONTAMINATED PASTURE

If the new introductions are still infected with GIN, they will be very resistant! This means it is important to dilute any eggs they may still be passing with "farm" parasites, which is accomplished by turning them onto contaminated pasture, ideally one which has a high level of refugia.

4.3.1 When no contaminated pasture is available

Keep the treated animal(s) in isolation. During the grazing season, have a FEC performed 14 days after treatment. If more than 10 animals, samples can be pooled from 10 randomly selected animals. If fewer, do individual samples. If still positive, consult your flock veterinarian on other available treatments. If the animals are purchased during the winter months, FEC may not be useful as the parasites are hypobiotic (arrested). A FEC should be done in the spring prior to turnout to assess if they are still infected with resistant GIN.

★ 5. INVESTIGATE TREATMENT FAILURE

5.1 IS IT PARASITES THAT ARE MAKING THE ANIMALS SICK?

If the sheep/goats appear not to respond to treatment or are showing signs of parasitism despite deworming recently, the reason for this must be investigated. Other diseases may be the cause of poor growth or diarrhea, or even sudden death. Poor growth can be nutritional in origin (e.g. poor pasture, selenium deficiency). Diarrhea can be due to coccidiosis. Pulpy kidney (*Clostridium perfringens* type D) can be the cause of sudden death on lush, green pasture. Anaemia may be due to liver flukes (*Fasciola hepatica*) although at this time in Canada, this parasite is not common.

Have FECs performed (if ML, CL or Startect were used, 14 days after treatment; if BZ was used 10 to 14 days after treatment). If animals have died, have your veterinarian or local diagnostic laboratory perform a necropsy and abomasal worm count. Just seeing a few worms in the stomach is not "proof positive" that they killed the animals; they need to be measured and counted as described previously.

5.2 TESTING FOR PRESENCE OF ANTHELMINTIC RESISTANCE

If the FEC is still high, perform a drench response test as described previously. Make sure you are delivering a sufficient dose of the dewormer. If the treatment fails to reduce the FEC, you and your veterinarian need to discuss if you should pursue a faecal egg count reduction test. If AR is confirmed, review this document, and with your flock veterinarian develop a plan for managing parasites.

5.3 RE-ESTABLISHING A SUSCEPTIBLE PARASITE REFUGIA – CAN IT BE DONE?

If AR has been identified on a sheep property, is it possible to re-establish susceptible refugia? It may be possible but it isn't easy. The following has been recommended: 1) the refugia is reduced through either leaving the pasture fallow for a long period of time, grazing with another species such as cattle or horses (not goats or sheep), or ploughing and reseeding; 2) lambs or kids that have been purposely infected with susceptible GIN are then introduced to seed the pasture with susceptible L3; 3) then the flock of that farm is grazed on these pastures and the new infection will dilute the level of resistant parasites carried by those animals. Difficulties with this approach are to first invest in reducing the refugia and then to locate lambs or kids with heavy loads of susceptible GIN.

PARASITE CONTROL ON ORGANIC SHEEP / GOAT FARMS

Sheep and goats raised organically need not suffer from clinical parasitism, but producers must invest heavily in the principles of sustainable Integrated Parasite Management. A recent study performed on conventional and organic sheep farms in Ontario and Quebec found little difference in the level of parasites on these two types of farms, although there was tremendous variation in parasite loads between individual farms (abstract in Appendix 5). Producers must remember though that reserving treatment until animals are clinically diseased is not appropriate for proper control of GIN parasitism and has welfare implications. Use of alternative compounds should be avoided without scientific evidence of efficacy and safety, as this will worsen the problem. Below are the most recent regulations on organic production of livestock with respect to internal parasite management (updated 2018).

The Organic Production Systems General Principles and Management Standards[2]

(CAN/CGSB 32.310-2015 Amended March 2018)

Section 6.6 Livestock health care

6.6.11 Organic livestock operations shall have a comprehensive plan to minimize parasite problems. The plan shall include preventative measures, such as pasture management, fecal monitoring and emergency measures in the event of a parasite outbreak. By way of derogation, if preventative measures fail, due to climatic conditions for example, or other uncontrollable factors, the operator may use parasiticides that are not listed in Table 5.3 of CAN/CGSB-32.311, provided that:

a) observation of the animal or fecal test results, as appropriate for the species, indicate that livestock is infected with parasites;
b) the operator has written instructions from a veterinarian indicating the product and method to be used;
c) withdrawal times are twice the label requirement or 14 days, whichever is longer;
d) meat animals less than 12 months old receive only one treatment. Older meat animals shall receive a maximum of two treatments. Meat animals that require additional treatment shall lose their organic status;
e) dairy animals that receive more than two treatments in a 12-month period, whether of parasiticides, antibiotics or one of each, shall lose their organic status and go through a 12-month transition period. Meat animals that receive more than two treatments of parasiticides shall never be organic;
f) a dam may be treated during gestation;
g) laying hens that receive more than one treatment in a 12-month period shall lose their organic status. Treatment of the flock, rather than individual hens, is permitted;
h) the operator provides a written action plan, with a timeline, describing how they will amend their parasite control plan, to avoid similar emergencies.

6.6.12 Poultry or breeding livestock treated with a parasiticide or veterinary drug not listed in Table 5.3 of CAN/CGSB-32.311 shall be considered non-organic meat animals. Exceptions pertaining to parasiticide use may apply (see 6.6.11).

ORGANIC PRODUCTION SYSTEMS PERMITTED SUBSTANCES LIST[3]

(CAN/CGSB-32.311-2015 Amended March 2018)

Table 5.3 – Health care products and production aids

Parasiticides and antimicrobials	Shall respect requirements set out in 6.6 of CAN/CGSB-32.310 with regard to the use of internal parasiticides.

For more information, consult the entire documents.

OTHER IMPORTANT NEMATODE PARASITES

Below are descriptions of other important nematode (roundworm) parasites - which can cause significant disease but less commonly than those mentioned above.

SMALL INTESTINE

COOPERIA CURTICEI

This parasite infects both sheep and goats. It is small, 0.5 to 0.8 cm. Eggs are typical GIN type. The life cycle is typical with the larvae burrowing into the intestinal crypts and adults living on the surface. Prepatent period is 2 weeks. *Cooperia* becomes hypobiotic (arrested) in the late fall. Lambs/kids at pasture are most likely to develop heavy infections. Adults tend to remain immune but shed low numbers of eggs. Signs are mild or absent unless infection is

2 http://publications.gc.ca/collections/collection_2018/ongc-cgsb/P29-32-310-2018-eng.pdf
3 http://publications.gc.ca/collections/collection_2018/ongc-cgsb/P29-32-311-2018-eng.pdf

very heavy in which case poor appetite and growth are most notable. Parasite numbers may be high, even without severe signs of disease. The parasite causes villous atrophy of the intestine. In cattle studies, AR develops quickly in *Cooperia*.

BUNOSTOMUM TRIGONOCEPHALUM

This parasite is also known as the "**hookworm**". It infects both sheep and goats. It is fairly large at 1 to 3 cm in size and the adults live in the small intestine. Eggs are typical GIN type. At this time, there is no evidence of this parasite in central Canada, so it is likely uncommon. The infective L3 can penetrate the skin where they migrate to the lungs and then migrate to the digestive tract. Ingested L3 do not migrate to the lungs. The prepatent period is 4 to 8 weeks. This parasite prefers more tropical climates. Adult hookworms suck blood and a fairly low infection of 500 worms is associated with anaemia, hypoproteinemia (bottle jaw), weight loss and death. The carcass is pale. The intestinal lining is reddened and edematous. The worms may be seen attached to the intestinal mucosa or in the lumen of the intestine.

STRONGYLOIDES PAPILLOSUS

This parasite is also known as the **"threadworm"**. It infects sheep and goats as well as other ruminants. The worms are very slender < 1 cm in length. The eggs are larvated and are about 50% as large as typical GIN eggs. It is very commonly found in diagnostic samples. Only the females have a parasitic stage and both females and males are free-living. The females can produce eggs by parthenogenesis (asexual reproduction). To build up significant infestations in the environment, the conditions must remain warm and moist, as the larval stages are all susceptible to environmental conditions. L3 infective larvae can be ingested, penetrate skin or infect lambs /kids through dam's milk. The prepatent period is 8 to 14 days. While eggs are commonly seen on faecal egg counts, disease is typically uncommon and not severe. In very high levels of infection, young lambs/kids (2 weeks of age) may exhibit signs of diarrhea and reduced gains. High faecal egg counts are not always indicative of a clinically significant infection. The adult parasite can cause inflammation of the intestine and villous atrophy.

LARGE INTESTINE

CHABERTIA OVINA

This parasite is also known as the **"large-mouthed bowel worm"** and is not common in Canada. It infects both sheep and goats. The adults are 1.5 to 2 cm in length and found in the colon. Eggs are typical GIN type. As few as 300 adult worms can cause disease. The adults ingest chunks of the lining of the colon and cause loss of blood and protein. The L3 can over-winter on pasture and the L4 can become hypobiotic in the large intestine and re-emerge in the spring. However, severe disease is unusual in temperate climates. Severe infections cause diarrhea with blood and mucus, sometimes with visible worms in the feces. The lamb/kid develops anaemia, hypoproteinemia and weight loss. The young larvae are in the caecum and the adults in the colon. The damaged mucosa is evident along with visible worms.

OESOPHAGOSTOMUM COLUMBIANUM

This parasite infects sheep and goats and wild ruminants. It is also known as the **"nodular worm"**. The adult worms are slightly > 1 cm in length and found in the large intestine. Eggs are GIN type. It is considered an important parasite in tropical and sub-tropical countries but is found worldwide. The L3 penetrate the mucosa of the small or large intestine and form nodules where they develop to the L4 stage. They may remain in the nodules for up to 1 year. When the L4 emerge, considerable damage may be caused to the intestinal wall. The prepatent period is 45 days. Severe infections are typified by dark, green diarrhea; milder infections by intermittent diarrhea and poor growth. The nodules are found in the lower intestine and can be up to 2 cm in diameter. The inflammation associated with rupture of the nodules can cause adhesions and even perforation resulting in peritonitis.

TRICHURIS OVIS

This parasite infects sheep and goats and occasionally other ruminants. It is also called **"whipworm"**. The adults are very long worms (4 to 8 cm) with a thick posterior and very slender anterior which is usually buried in the mucosa of the large intestine. Eggs are oval and brown with a transparent plug at either end. The L1 remains in the egg in the environment and is the infective stage. Eggs can remain viable in the environment for up to 4 years. Once ingested, the caps on the eggs are digested, releasing the larvae, which then penetrates the lining of the distal small intestine and

large intestine. The prepatent period is 7 to 10 weeks. Infections are quite common in central Canada but significant disease is not. Most infections are light and there are few clinical signs. The parasite causes a mild colitis. The adult parasite is easily seen on postmortem. The lining of the colon is haemorrhagic around where the worm is imbedded.

LUNG

DICTYOCAULUS FILARIA

This parasite infects sheep, goats and deer and is more commonly known as "**lungworm**". The adult worms live in the large and smaller airways (bronchi) of the lungs and are quite large (5 to 10 cm). While described as being found worldwide, infections are sporadic and uncommon in Canada. The females lay eggs in the airways, which are coughed up and swallowed. The eggs hatch and the L1 larvae are passed in the feces where they moult to L3. A routine faecal examination may miss them because the eggs have hatched - a special test (Baermann technique) is needed to diagnose an infection. Larvae can over-winter on pasture but most pasture contamination comes from infected sheep and goats in the same grazing season. Coughing (with mucus) and ill-thrift are the most common signs. Secondary pneumonia can exacerbate the signs. The lungs are wet and red and the bronchi, and potentially trachea, are filled with worms.

MUELLERIUS CAPILLARIS

This very common parasite infects sheep, goats and deer. It is also called **"nodular lungworm"**. Although 1 to 3 cm in length, these worms are hard to see because they are located in the lung tissue and not the bronchi. The life cycle requires an intermediate host, in this case snails and slugs. The L1 larvae are coughed up and swallowed, then passed in the faeces. They penetrate the foot of the mollusc, mature and reside there for 2 to 3 weeks until inadvertently eaten by a sheep or goat when grazing. The mollusc is digested releasing the L3 larva, which penetrates the digestive tract and travels through the blood and lymphatic system to the lungs. The prepatent period is 6 to 10 weeks. The adult worm can live for several years, with the result that infection and lung damage from the infection can be cumulative. This parasite is not considered to be very pathogenic in sheep, but it is in goats. Coughing, difficult breathing and pneumonia can be seen. Radiographs reveal increased density (white) dorsally in the lung suggesting disease due to *Muellerius* and / or caprine arthritis encephalitis (CAE). The dorsal surface of the lung has nodules from very small up to 2 centimetres in diameter. The nodules are hard (lead shot) and contain one to several worms. When the worm dies, the nodules become necrotic. More extensive areas of lung consolidation occur in goats. Treatment is difficult because of the scarring of the lung and may need to be performed more than once.

NERVOUS SYSTEM

PARELAPHOSTRONGYLUS TENUIS

The final (definitive) host is the white-tailed deer, but many other ruminants can be infected - in particular, sheep, goats, moose and new world camelids (llamas, alpacas). This parasite is more commonly known as **"deer meningeal worm"**. The disease caused by this worm is also known as "**moose sickness**". These are large, slender worms up to 9 cm in length. The intermediate hosts are snails and slugs. Normally, when deer ingest an infected snail or slug, the worm migrates up the spinal nerves from the gut - eventually reaching the brain and spinal cord. Eggs are laid in the small blood vessels and make their way to the lungs where they hatch, and larvae are coughed up, swallowed and passed in the faeces.

It is difficult to control this disease as the parasite is common in white-tailed deer, and the snails and slugs are everywhere. Preventing the sharing of pastures is very difficult in Ontario and Quebec where white-tailed deer are very common. The infection in white-tailed deer doesn't cause disease, but in non-target ruminants, severe neurological signs may occur. This is because of aberrant (misdirected) migration in the non-target host. The worms migrate down trunk nerves or into the brain causing inflammation. The animal becomes disorientated and may develop severe pruritus (itching) along the trunk nerves. Moose may wander into towns, staggering. Sheep and goats may develop paralysis - particularly in the hind-end, circling, blindness, weight loss and death. Treatment often involves long-term use of anthelmintics (e.g. fenbendazole) as well as anti-inflammatory drugs. On postmortem, the worm may be identified in the brain or spinal canal. In Ontario, most aberrant infections are seen in alpacas and llamas but may not be recognized in sheep or goats without a postmortem diagnosis.

NON-NEMATODE INTERNAL PARASITES

There are many other types of internal parasites that cause significant disease in sheep in central Canada. Below includes descriptions of three types: protozoa, which includes coccidia (*Eimeria*) and *Cryptosporidium*; cestodes, both adult tapeworms and larval (intermediate stage) tapeworms; and liver flukes. The lifecycles are described under each parasite.

PROTOZOA

CRYPTOSPORIDIUM PARVUM

The parasite infects sheep, cattle, goats, horses, deer and humans. The disease is known as **"cryptosporidiosis"** or **"crypto".** It is a microscopic protozoal (one-cell) parasite of the small intestine. It is zoonotic and may cause severe gastrointestinal disease in people that can last for well over a week. It has a similar life cycle to *Eimeria* spp (coccidia) but it takes only a few days to complete the life cycle in animals and produce oocysts (eggs). The oocysts are infective in fresh faeces, are very resistant to environmental factors, and build-up quickly in lamb / kid rearing areas. The animals may also auto-infect, so disease may be severe without high levels of environmental contamination. The main sign is diarrhea in lambs/kids, as young as a few days of age but up to 3-4 weeks. Affected lambs/kids can become very unthrifty because of loss of the ability of the small intestine to absorb nutrients, i.e. villous atrophy so even if milk is fed, it may not be digested or absorbed properly. Additionally, fluid is lost. They can become severely dehydrated, depressed and very thin, and in some outbreaks this parasite can be a significant cause of lamb/kid death. There is no effective treatment available in Canada although halofuginone (Halocur®, Merck Animal Health) has been tried in severe cases. Decoquinate has been used as a preventative although it is not licensed for this use. Speak to your veterinarian about whether either of these drugs should be used in your animals. *Cryptosporidium* also infects humans so great care should be used when handling sick lambs or kids. To properly diagnose, a faecal sample should be submitted to an animal health diagnostic laboratory for analysis with a specific request to check for this parasite. Lesions on postmortem are mild and usually only seen in the ileum of the small intestine.

COCCIDIOSIS CAUSED BY *EIMERIA*

Coccidia are microscopic, protozoal parasites of the intestine and occasionally liver. The pathogenic species are responsible for one of the most important parasitic diseases of youngstock in Canada and in the world. It can occur on pasture or in the barn. Severe coccidiosis can occur at any time of the year. The different species of *Eimeria* are host-specific, i.e. sheep coccidia only affect sheep and goat coccidia, goats. None cause disease in humans.

Table 3. Severely pathogenic species of *Eimeria* in sheep and goats

Species	Coccidia Name	Prepatent Period	Pathogenicity
Sheep	*Eimeria crandallis*	15 to 20 days	++
Sheep	*Eimeria ovinoidalis*	12 to 15 days	+++
Goat	*Eimeria arloingi*	20 days	++
Goat	*Eimeria christenseni*	14 to 23 days	++
Goat	*Eimeria ninakohlyakimovae*	10 to 13 days	+++
Goat	*Eimeria caprina*	17 to 20 days	++

Some species are mildly disease causing and others cause very serious and even fatal disease. The difference is in how widespread in the intestine, and where, the coccidia infect the cells. The small intestine has an amazing ability to recover from damage but not so the large intestine. If only the small intestine is affected, the goat/sheep suffers much less. The life cycle of the coccidia is quite complicated as can be seen from Figure 4. One coccidial oocyst can result in up to 50 million intestinal cells being destroyed. The act of schizogony (asexual reproduction) and gametogony (sexual reproduction) causes the cells of the intestine to rupture and release the next stage of the life cycle. It is important to understand the basics of this life cycle in order to understand how to best control the infection.

Coccidiosis – The disease

This is a disease that most often affects youngstock – either nursing or weaned, with diarrhea and poor growth. Adult sheep do not exhibit disease and only very rarely do adult goats. Coccidiosis is often seen along with other diseases, most often pneumonia and soremouth (orf). We also more often find coccidiosis as a herd/flock problem when there are other stresses in the herd/flock, for example crowded conditions or weather stresses (e.g. cold, heat, high humidity).

Acute severe coccidiosis

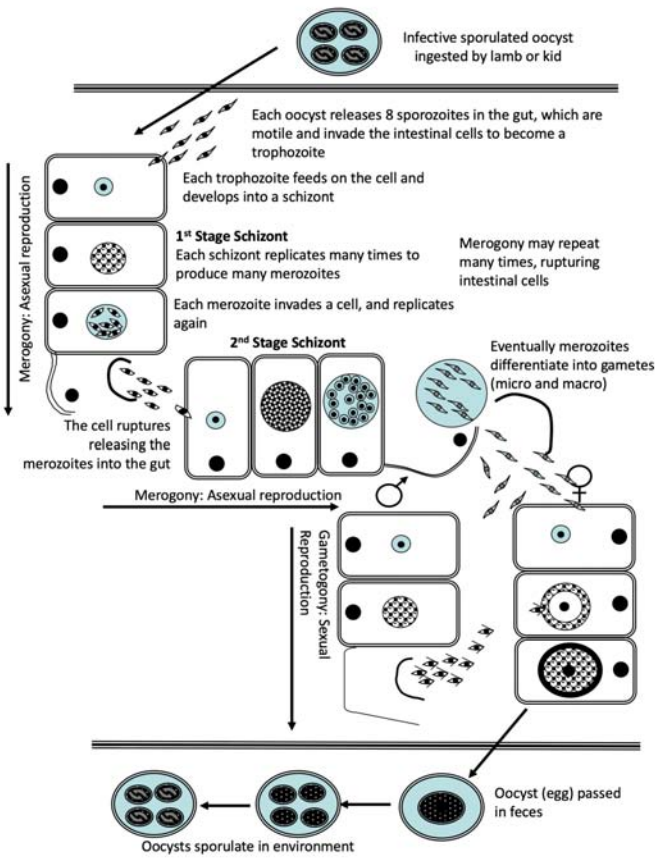
Kids / lambs are very ill and many may die without prompt and appropriate treatment. They may be affected as early as 3 weeks of age but mostly 5 weeks on – depending on how contaminated the environment is with oocysts. Signs may appear fairly suddenly and a kid/lamb only mildly ill the day before, may be very sick the next day. Diarrhea is an important feature and may be watery and brown or may have blood in the stool (black and tarry and / or red streaks of fresh-appearing blood). They are dehydrated and often anaemic. They will invariably be depressed but fever is not always present. Some animals may strain from the inflammation of the lower large intestine and pass only watery blood. In such an outbreak, it is common to have deaths.

Chronic coccidiosis

These animals may have had acute severe coccidiosis earlier or may not ever have been noticed ill. The affected group appears unthrifty and grows more slowly. They are thin, pot-bellied and small – although their heads may continue to grow giving them a runty appearance. The hind end may be dirty (daggy) due to the soft stools and intermittent diarrhea. Kids and lambs with chronic coccidiosis may never fully recover from the effects of the disease and are stunted their entire lives.

Age at which coccidiosis is seen

Figure 4. Lifecycle of the coccidia



Coccidiosis is a disease of young kids and lambs. The most common age to be affected is 4 weeks to 5 months. Nursing lambs/kids appear more at risk of acute severe coccidiosis. Occasionally, kids or lambs as young as 3 weeks may be affected–diarrhea younger than this is more often due to agents of neonatal diarrhea (rotavirus, coronavirus, *E. coli* and *Cryptosporidium*). If older animals appear to be suffering from chronic coccidiosis, it may be the lasting effects from an infection from when they were younger. It is important to remember that shedding of the coccidia eggs – oocysts – is not evidence of disease, but only evidence of infection. Adult goats and sheep often shed oocysts particularly in the peripartum period, many of which may be from non-pathogenic *Eimeria*. It is also important to remember that severe disease can occur before infection is far enough advanced for the sheep/goat to be shedding oocysts.

How the damage is done

A single oocyst, once ingested, releases sporozoites that swim through the contents of the intestine and then each penetrate a cell on the lining of the intestine. Inside the cell, the sporozoite becomes a trophozoite and feeds until large enough to become a schizont. This schizont divides many times within the cell to produce up to 1,000 merozoites. The cell then

ruptures, releasing these merozoites that again swim through the intestinal contents. This asexual reproduction is called merogony. Each merozoite penetrates another intestinal cell and goes through another cycle again releasing thousands of merozoites. Eventually, the merozoites that are released infect intestinal cells and change into either male (micro) or female (macro) gamonts. Male microgamonts divide many times, release male cells that fertilize the female cells (sexual reproduction or gametogony), and in each infected intestinal cell, an oocyst or egg is formed. These oocysts burst out of the cell and are passed in the feces.

There are many stages, and at each stage intestinal cells are invaded and then destroyed – causing repeated damage to the intestine. The intestinal damage can release blood and cause inflammation of the lining of the gut. The animal loses blood, water and protein and cannot absorb nutrients as efficiently. If enough damage is done, it becomes extremely ill and may die.

Diagnosing coccidiosis

The best way to diagnose coccidiosis is based on the clinical signs in the group of animals as described above, and evidence of infection based on postmortem if any animals have died. Taking a fecal sample and having a quantitative count of the number of oocysts (fecal oocyst count or FOC) in the stool can also be helpful but there are many ways it can be misinterpreted so caution must be used.

A low FOC does not rule out coccidiosis. Acute disease may be present before the prepatent period is reached – so the FOC may be very low. Diarrhea will dilute the FOC. A high FOC (even in the thousands) can occur when the kid or lamb is infected with a low pathogenic species. While it is possible to differentiate the species of coccidia based on the microscopic appearance of the oocysts, it is difficult and must be done by a trained parasitology technician using special techniques. Additionally, a moderately high FOC will often be present even long after the animal develops immunity, and may rise if the animal's immune system is stressed – all without it suffering from the disease.

Factors affecting if coccidiosis is a problem in the herd or flock

Why does one herd/flock have a problem with coccidiosis and another does not? Why are some years, or some times of the year worse than others? We need to consider that presence of the disease agent alone is often not sufficient for a coccidiosis problem to occur, but that different factors all play a role. It is easier to consider the factors in three categories:

The Parasite

What species of coccidia are present and how pathogenic are they? How many oocysts are present in the environment and where are the oocysts? Are they contaminating places that allow for easier transmission to the kids/lambs?

The Animal

How immune is the kid or lamb? Younger animals are more susceptible. Are the kids/lambs ill with another disease that could weaken their immune systems, e.g. soremouth or pneumonia? Have they been stressed by changes in the diet or a poor diet, by crowded conditions or bad weather? Have groups been mixed (e.g. younger moved in with older)? Has there been fighting and competition at the feeders? Have they recently been weaned?

The Environment

The oocysts must mature (sporulate) before they are infective. Time to sporulation depends on moisture, oxygen (e.g. exposed to the air rather than buried in the bedding pack) and temperature. Exposure to sunlight will assist killing of the oocysts so pasture tends to be safer than indoor housing. Animals raised on dirty bedding are more at risk than those on slatted floors or clean bedding. Type of feeders and waters may have an effect if they are designed to prevent contamination with manure. High stocking densities, build-up over the kidding/lambing season – all increase the load of oocysts in the environment and thus increase risk of disease developing.

Controlling coccidiosis

Reducing stress to the lambs / kids

Keep stocking densities low. Make sure there is adequate space, not just for the dams but also the offspring. 2.5 square meters per nursing pair is not unreasonable and 3 square meters of pen space is preferred. Make sure that ventilation is adequate to prevent build-up of humidity and ammonia levels in the barn. Avoid drafts and daily temperature fluctuations. If outdoors, make sure they have shelter from inclement weather including hot sunlight (e.g. a run-in shed). Nutritional stresses may be from artificial rearing with poor quality or poorly managed milk replacer, nutritional deficiencies from a poor diet (e.g. poorly digestible forages, inadequate vitamin E and selenium, other mineral deficiencies). Other diseases such as soremouth (orf) and pneumonia may be worse under stressful conditions. In pastured kids and lambs, concurrent infection with gastrointestinal nematodes may increase the risk of disease from coccidia. Infections can be managed through the judicious and proper use of prophylactic anti-coccidial medications. Kids or lambs that are ill should be promptly treated to prevent more contamination of the environment.

Reduce risks from the environment

The biggest source of contamination of the environment is from kids or lambs with uncontrolled infection. One oocyst eaten by a kid or lamb will result in 10,000 new oocysts produced and there can be thousands to millions of oocysts excreted per gram of feces (30 grams = 1 ounce). These oocysts can survive in the barn for many months. Outbreaks of coccidiosis late in the kidding/lambing season often occur because of the build-up through the winter, particularly if the pens have not been cleaned. Forage and grain feeders need to be designed so that manure contamination from defecation or dirty feet is minimized. Bedding should also be kept fresh and dry.

Oocysts are very resistant to desiccation (drying) and many disinfectants. Sunlight will help to kill oocysts on pasture. They will sporulate (become infective) in as little as 2-5 days at temperatures as low as 12° C so potential for environmental build-up is massive.

To disinfect: Remove all bedding, old feed, water and manure first by scraping. If possible, one should steam-clean the entire pen and equipment. The extreme heat will help to kill the oocysts as well as physically remove them (they are sticky!). Examples of temperatures needed to kill oocysts: At 56° C for 1 minute; at 54° C for 8 minutes; and at 50° C for 150 minutes is required to kill 100% of the *E. arloingi* oocysts of lambs. Lower temperatures can take as long as 24 h to kill the oocysts. Few disinfectants are effective against oocysts – check the label. Ammonium based disinfectants may be most effective but surfaces need to be initially cleaned. OO-Cide (Vétoquinol Canada Inc.) (ammonium chloride and sodium hydroxide) can be used in empty barns after all organic matter is removed. Follow directions explicitly to remain safe.

Reducing risk from adults

Adults are not an important source of oocysts except possibly in the periparturient period, when shedding increases as with GIN parasites. To control this, some give medicated feed to the adults during this period. The decision to do so should be made with your flock/herd veterinarian's advice as not all situations warrant this practice and this practice alone will likely not prevent coccidiosis in the offspring. It is certainly not a replacement for control strategies in the youngstock both before and after weaning.

Use of anti-coccidial drugs

The use of preventive anti-coccidial medications (also called coccidiostats) is a common method to control coccidiosis in sheep and goats. However there are some issues.

- There is only one approved medication for goats in Canada (monensin); all others require a veterinary prescription.
- Using these drugs requires more exacting feeding management to make sure the individual kid/lamb gets the correct amount every day.
- It also requires getting the medication into the very young animal (perhaps even less than one week of age) in sufficient quantities to prevent disease.
- Even the best drug cannot protect very stressed animals or those in a heavy contaminated environment.
- Coccidia can and do develop resistance to these drugs so investigate treatment failure.

- For Certified Organic farms, drugs cannot be added to the feed or water and meat animals can be treated only once if they become clinically ill (i.e. no prophylactic use).

The goal of using these medications is to control the level of infection so as to prevent the disease but to still allow enough infection so that the young animal develops immunity. To do that, usually the drug needs to be effective from birth to 3 - 4 months of age. It is usually delivered in creep feed (i.e. a grain mix delivered only to the youngstock) to assure sufficient intake and not overdose. To make sure the animals eat it, it needs to be palatable, not settle or sort out of the mix and there needs to be plenty of trough space to make sure all animals can consume what they need.

There is evidence that development of immunity to one *Eimeria* species does not mean that the lamb / kid is immune to all species; immunity seems to be species specific. This means that treatment might need to continue through different areas of the barn or pastures until immunity has developed to all important species. Work is ongoing at the University of Guelph to determine more on this.

The following medications can be used to control coccidiosis in lambs and kids. If not licensed for use in a particular species (sheep versus goats) or class (lambs versus adults), a veterinary prescription needs to be written.

Lasalocid

Lasalocid (Bovatec® / Avatec®, Zoetis Canada), an ionophore antibiotic, is licensed for use in lambs but not kids. To use this product in kids, a veterinarian must write a prescription for the feed company. It kills the "free living" stages of the coccidia (sporozoites and merozoites) as they move from cell to cell in the intestine (see Figure 4). Because it kills the coccidia, it may help control disease after the animal is infected. In lambs it is approved as a feed additive to be fed free choice, at a concentration of 36 ppm = 36 mg of drug per kg of feed. To be effective a kid/lamb needs to consume 1 mg lasalocid /kg body weight per day. To determine if they are consuming enough, weigh feed consumed daily, weigh the animals and calculate what they are eating. E.g. a 10 kg (22 lb) kid must eat 0.28 kg of creep daily (at 36 ppm of lasalocid) to receive a therapeutic dose. Lambs and kids must be introduced to a ration slowly over 2-3 weeks to acclimate the rumen organisms. Meat withdrawal is 2 days. It is not to be used in lactating dairy animals.

Monensin

Monensin (Rumensin™, Elanco Canada), another ionophore antibiotic, was very recently licensed for sheep and goats in Canada (11 and 22 gm/tonne). It works the same as lasalocid. Generally, suggested feeding rates are 11 gm/tonne or ppm free choice feed and 22 ppm limit fed. Monensin has a narrower safety margin than lasalocid. The LD 50 (kills 50% of the animals that ingest at this level) for sheep is 11.9-mg/kg bw/day and for goats is 26.4-mg/kg bw/day. The level that kills 1% is much lower so caution must be used to prevent animals from ingesting too much. Feed refusal and stiffness, as well as death may occur if the levels are too high in the feed. Lambs and kids must be introduced to a ration slowly over 2-3 weeks to acclimate the rumen organisms.

Decoquinate

Decoquinate (Deccox® 6% Premix, Zoetis Canada) is not an antibiotic nor will it improve feed efficiency. It is licensed for sheep but not goats in Canada. The recommended therapeutic dosage is 0.5 mg/kg bw daily for sheep and goats although there is strong evidence that 1 mg/kg bw is much more effective in these species. The inclusion rate in a complete feed is 1.67 kg (when feeding rate is 50 g / 10 kg bw) or 3.34 kg (when feeding rate is 25 g/ 10 kg bw) of 6% premix per tonne of feed (100 and 200 g/tonne of decoquinate respectively). Intakes can be low in nursing lambs; it is recommended to go with the higher inclusion rate prior to weaning.

Decoquinate works very early in the life cycle, killing only the sporozoites as they first infect the kid or lamb, and so is not effective to treat coccidiosis (see Figure 4). The recommended feeding period is a minimum of 28 days. However, because it is so effective early in the cycle, for the kid or lamb to develop immunity, it should be treated for a minimum of 3 cycles (e.g. 70 days) and perhaps longer. There is no meat withdrawal period for this product. It is not to be used in lactating dairy animals.

Decoquinate is available in another form as an additive to milk or milk replacer for kids being raised artificially (Deccox M 0.8%). It is not licensed for use in lambs or kids. It contains 8 g decoquinate / kg premix. The amount added to the milk will depend on whether the milk is limit or free choice fed. E.g. 10 kg kid needs 5-10 mg/day.

However, it is important to remember: the milk must be agitated for 5 minutes before feeding as well as during feeding to prevent settling out and under dosing, so only use to feed individuals (e.g. don't use with a kid bar).

Toltrazuril

Toltrazuril (Baycox® 5% Bayer Animal Health) is licensed to control coccidiosis in lambs, piglets and calves in Canada. It is a drench to be given at a very specific time of the animal's life. The lamb dosage is 20 mg/kg bw (1 ml/2.5 kg bw) *once* prior to the first expected onset of disease. In many flocks this will be ~ 3-4 weeks of age but should be farm specific. It has great persistency, close to 20 days. The meat withdrawal is 48 days. It must NEVER be used in lactating dairy goats or sheep. Studies in other countries have found it is efficacious in kids, usually at 20 mg/kg bw (same dosage as lambs). Because its use in kids is extra label, meat withdrawal should be longer. Consult CgFARAD to obtain a withdrawal period.

Toltrazuril kills coccidia in the intracellular stages including merozoites and micro- and macrogametes. This means that it is very effective in reducing any infection in the animal when they are treated. Additionally, there is persistency of the drug reducing the need for retreatment. Because of how it works, toltrazuril must be used very differently from other anti-coccidial drugs. But there are several factors that must be considered before deciding that this is the method to control coccidiosis in your herd/flock:

- All individual lambs/kids must be treated. Even leaving one untreated animal can reinfect the remaining animals by increasing the environmental contamination.
- The animals must be at the correct age. Treatment is done around one week prior to the first time coccidiosis is seen in your lambs/kids. On most farms that is 4 to 5 weeks of age. This often means treating each animal when it reaches 3 to 4 weeks of age. This means you can't treat the group at one time but rather when the individual animal reaches that age. Waiting until everybody is old enough means that the older animals may be diseased and are shedding large numbers of oocysts, thus increasing the environmental contamination, which can overwhelm the drug.
- Some producers wish to treat lambs when they are very young, the same as the recommendation in piglets (3-4 days of age). The reason given by these producers is that a lower dose is needed and a cost saving is perceived. It may also require less labour. Keep in mind that the coccidia that affect piglets, *Isospora* spp have a much earlier infection period than *Eimeria* spp in lambs. While the drug has persistency, it is effective for 20 days at best. If toltrazuril is given at 4 days of age, it will not be protective at 25 days of age, the time when most lambs are getting infected. If used at 3-4 days of age, it is very likely that a repeat dose will be required to control infection.
- To treat animals at the right age, you must have a very good identification system, excellent records and handling facilities – along with enough reliable help, so that it is easy to find, catch and treat the lambs/kids at the appropriate age.
- Because of the persistency of the drug, you can't use it for kids or lambs slaughtered at light weights. If a lamb is treated at 28 days of age + 48 day meat withdrawal, it cannot be marketed before 76 days of age. Kids should have a longer meat withdrawal (have your veterinarian contact CgFARAD).
- You must continue to monitor the animals for signs of coccidiosis. In situations where there is still a large environmental load of oocysts, it sometimes is necessary to retreat in 3 to 4 weeks.

Amprolium

Amprolium (Amprol 9.6%, Huvepharma AD) is not approved for sheep or goats in Canada. It is more effective as a treatment than as a control. Its mode of action is that it acts on second-generation schizonts and so kills the coccidia later in the life cycle, after they have already done some damage. However, it also interferes with thiamine uptake by the intestine and so overdosing or chronic use can cause thiamine (vitamin B1) deficiency, also called polioencephalomalacia. Resistance of coccidia to amprolium has been reported in goats.

There are many suggested dosages for kids and lambs but the following has been reported to work well as a treatment without causing problems: for kids 50 mg/kg per day for 5 days; for lambs the dosage is reported at 20 to 50 mg/kg bw/day for 5 days. Although the amprolium can be added to the water it is impossible to assure adequate intakes of all animals, particularly in nursing or sick animals, so it is highly recommended to drench individuals.

Sulfonamides

This group of antimicrobials are to be used for treatment only. There are several types with efficacy against coccidia: sulfamethazine, sulfaquinoxaline, and sulfadimethoxine are usually given as a drench or in feed or water. Toxicity is a real risk from overdose or long-term treatment and signs are depression and kidney failure. Chronic exposure to sulfa drugs will contribute to antimicrobial resistance. As with amprolium, resistance of coccidia to sulfa drugs has been reported in small ruminants.

Diclazuril

Diclazuril suitable for use in kids and lambs is not available in Canada. A product containing diclazuril, Clinacox 0.5% (Merck Animal Health) is sold as a feed additive for turkeys and poultry. Because the vehicle carrying the drug is very different in Clinacox (it was designed for a bird gut and not a ruminant gut) from the ruminant version we don't know if the drug is available to work in a lamb or kid intestinal tract. Like toltrazuril, it works against the intracellular forms of coccidia. In the European Union it is available as a sheep drench (Vecoxan, Elanco Animal Health). The label indicates a single administration of 1 mg diclazuril per kg/bw most commonly at about 6-8 weeks of age, or two administrations beginning at 3 to 4 weeks of age and the second about 3 weeks later. Like toltrazuril, it needs to be given early in disease to prevent damage. Unlike toltrazuril, it is not persistent and meat withdrawals are shorter.

Summary

Coccidiosis is a common cause of disease in kids and lambs and a very important internal parasite. Although environmental control must be part of the herd health approach to this disease, judicious use of anti-coccidial drugs may be necessary to ensure adequate control. Any coccidiosis control program should be designed with your flock veterinarian as each farm and its challenges are unique.

TAPEWORMS (CESTODES) OF SHEEP

All tapeworms require two hosts: the intermediate host where the larval tapeworm develops, and the final or definitive host where the adult tapeworm grows, feeds and produces eggs. The final host must eat all or part of the intermediate host to get infected.

MONIEZIA EXPANSA

Tapeworm segments in lamb faeces

This is the common tapeworm of sheep and goats. The adult is found in the small intestine. It is white and comprised of segments (egg packets) 1 to 1.5 cm wide, and a scolex (head). The head has suckers that it uses to hold onto the intestinal wall. It can be quite long (metres) with many segments. The eggs are triangular-diamond shaped and easily identified on faecal examination. Each egg contains one embryonic tapeworm. The intermediate host is a free-living forage mite. The eggs are passed in the faeces of the sheep and goat and the forage mite ingests them. The eggs then hatch and the larvae migrate to the body cavity of the mite where they develop into a cysticercoid (a tapeworm head in a solid structure). When the mites are ingested by sheep / goats while grazing, cysticercoids develop into adults. Ingestion to egg production in sheep takes about 6 weeks. Interestingly, the adult tapeworms do not live long - approximately 3 months. Infection is usually worse in summer months but the cysticeroids can overwinter in the mites.

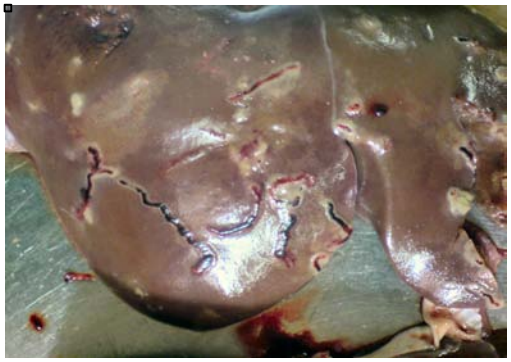
This tapeworm does not cause significant disease in sheep and goats but the segments are easily seen in the feces and cause the owner concern. However, a severe infection can be associated with diarrhea and unthriftiness, and very rarely the volume of parasites in the gut is associated with intestinal blockage and may be a risk factor for *Clostridium perfringens* Type D infection (pulpy kidney, also called enterotoxaemia). Lambs and kids develop immunity to the tapeworm and eventually the infection "self-cures". Treatment in Canada is limited to the use of BZs that have poor effectiveness against the parasite; the head of the parasite often survives the treatment.

THE INTERMEDIATE STAGE OF DOG TAPEWORMS

There are many types of dog tapeworms and some of these have an intermediate stage that can infect sheep. The damage they do to the sheep or goat is usually limited to the internal organs and / or carcass and the effect is usually

economic. However, these economic losses can be devastating with regards to losses associated with carcass condemnation. One of the types is also dangerously zoonotic. All can be controlled using similar measures.

ECHINOCOCCUS GRANULOSUS



Liver of lamb at slaughter with *T. hydatigena* bladder cysts and evidence of larval tracks.

Also known as "**hydatid disease**". This tapeworm of dogs and wild canids (coyotes and wolves) is highly zoonotic, i.e. causes disease in people. The tapeworm in canids is short (~ 0.6cm in length) making it difficult to see in the intestine or feces. The intermediate stage is a hydatid cyst. These cysts form in the liver or lungs of the sheep (or human) and can grow very large - up to 20 cm in diameter. Inside these cysts are tens to hundreds of tapeworm larvae (hydatid sand), often within brood cysts, each one capable of growing to a tapeworm. If the cyst forms in the abdomen, it may grow very large, containing several litres of fluid.

Many species of ruminants can be the intermediate host (along with humans) but sheep have been historically implicated in maintaining dog infections. The dog can be infected with thousands of adult tapeworms without signs and infected sheep rarely show signs. But humans with cysts develop signs of respiratory disease (lung) or liver disease, sometimes one or two decades after they are infected. If one ruptures, the person may die of shock. Children that play with infected dogs are particularly at risk. Dogs become infected because they are allowed to scavenge infected sheep carcasses. Evidence of cysticercosis (see below) in sheep indicates that the management of sheep and dogs is conducive to *Echinococcus granulosus* infection.

Most human cases in Canada are immigrants from countries with endemic hydatid disease or from northern Canada where the sylvatic cycle is likely moose and wolf/dog. *Echinococcus multilocularis* also occurs in Canadian canids and is traditionally also an Arctic region disease but recently has been shown to infect a high proportion of coyotes and foxes in Ontario and so poses a risk to dogs and people. It is not associated with sheep.

TAENIA HYDATIGENA

This is the definitive stage of the intermediate stage, *Cysticercus tenuicollis*. Dogs, wild canids (wolves, fox, coyotes), weasels and stoats are the final host (i.e. adult tapeworm occurs in these species). Sheep, goats, deer and cattle are the intermediate hosts. The dogs consume the infected intermediate host (e.g. sheep/goat offal) and become infected. The adult tapeworm sheds segments that contain thousands of eggs, in the feces. The eggs contaminate pasture or feed, which the sheep/goat eats.

The eggs hatch and the larvae migrate for about 4 weeks, eventually to the liver and abdominal cavity where each larva forms a cysticercus. This is a bladder-like structure that contains one embryonic tapeworm or protoscolex (head only). These cystic structures are fairly large (1 to 3 cm) but do not harm the sheep. Eventually the cysticercus will die and scar if not consumed. There are no clinical signs in the sheep/goats with the exception of a massive infection, which may cause liver failure. Infected dogs appear healthy as well.

Larval tracts, bladder-like cysts and scars can be seen in the liver, causing condemnation of that organ. While not economically devastating, the presence of this infection indicates a farm-level problem with management of deadstock (scavenging) or offal from slaughtered animals. This sets up the possibility of infection with *Taenia ovis* (see below), which can be economically devastating.

TAENIA OVIS

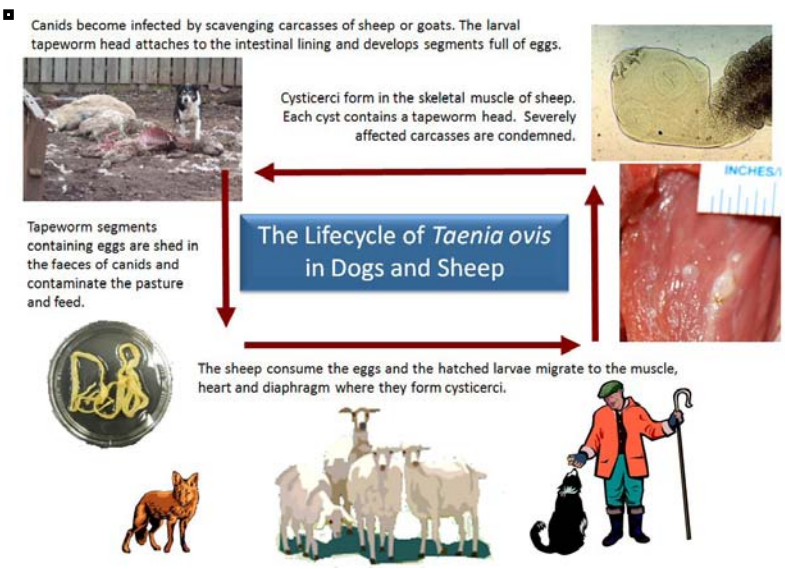
Also known as "**sheep measles**". The intermediate stage is *Cysticercus ovis*. The dog and wild canids (wolves, coyotes, foxes) are the definitive (final) host. Sheep and goats are the intermediate hosts (not deer). This is an emerging disease in Canada. The adult tapeworm is long and sheds segments that each contains over 70,000 eggs. The segments are found in the faeces but also on the coats of dogs and where the dog sleeps. Eggs have been known to disperse up to 80 metres across pasture, likely by insects or animal trampling. Sheep consume the eggs from contaminated pasture or feed.

The larvae migrate to the muscles (skeletal, heart, diaphragm, masseter muscles) where they form small cysts ~ 1 cm in size. After 2-3 months, the cysts are infective to dogs. This life cycle is portrayed in Figure 5. The cysts eventually die

and scar after a few months to a year, but the lesions are still present in the muscle. If a dog is allowed to eat an infective cyst, the prepatent period is 6 weeks. The biggest risk factor for this disease is improper disposal of deadstock allowing dogs to scavenge carcasses. There are no clinical signs in the sheep or the dog.

The cysts are apparent at slaughter and depending on the number and distribution, can cause the carcass to be either trimmed or condemned. The disease is not zoonotic, but affects meat quality so an outbreak - causing condemnation of a large percentage of carcasses - can be economically devastating to the industry.

Figure 5



Control and prevention of *Taenia ovis*

Control of the infection in sheep is done by controlling the infection in the farm dogs and preventing infection of coyotes, wolves and foxes. Once a sheep is exposed to the tapeworm eggs, there is no available method of preventing development of the cysts either through medication or vaccination.

Deadstock management

Proper deadstock management must be done to prevent scavenging of carcasses by all canids including livestock guardian dogs, neighbour's dogs and wild canids. In a study conducted in Canada, deadstock disposal was the most important aspect of whether a flock had a lamb condemned to *T. ovis*, with natural disposal, i.e. leaving the carcass to be scavenged increasing the risk 11 times! The second most important factor was producers reporting seeing their dogs scavenging sheep carcasses. If carcasses were properly composted or buried, the risk was much less.

Routine deworming of the dog

All farm dogs with access to the sheep and goats should be dewormed every month. The deworming schedule needs to be shorter than the prepatent period of the parasite, which is 35 days and is not associated with development of anthelmintic resistance. This is the program promoted for routine use in New Zealand (Ovis Management Ltd).

The deworming product must be effective against tapeworms; the products can only be obtained by veterinary prescription. Products containing either praziquantel or epsiprantel (Interceptor Plus™, Elanco Canada Ltd; Dolpac®, Vetoquinol; Drontal® Plus, Bayer; Cestex®, Zoetis; Droncit® Cesticide Tablets or Injectable, Bayer) or nitroscanate (Lopatol™, Elanco, Novartis) are the only ones that are effective. Consult your veterinarian for the best approach to this preventive treatment regime.

If sheep carcasses are to be fed to dogs

Feeding livestock guardian dogs can be expensive. Sheep that are otherwise healthy, dying of misadventure or cull animals that are euthanized without the use of a barbiturate, can be made suitable to feed to dogs. The cysts will die if the carcass is frozen to -10° C or colder for 7 days or longer, OR cooked to an internal temperature of 72° C.

Infection from wild canids (coyotes, foxes)

Usually wild canids do not defecate on pasture but rather just off trails, or camp (rest) on pastures as this is considered a high-risk activity for them although this may vary from farm-to-farm. However, when round bales are left on pasture to be foraged by pastured livestock, coyotes are often seen using them as lookouts or places of rest; this means there is a risk of feed contamination from wild canids. Because it is not possible to treat coyotes, nor convenient to not supplement forage on pastures, then it becomes even more important to prevent predation and deadstock scavenging by these animals. However, the risk from wild canids is overall much smaller than the risk from domestic dogs, whether working or guardian dogs, or pets if allowed to scavenge dead sheep.

LIVER FLUKES (TREMATODES)

FASCIOLA HEPATICA

The definitive hosts are sheep, goats, cattle, horses and deer. It is better known by the term "**liver fluke**". This is a parasite of the liver and bile ducts and is leaf-shaped and flat, about 2.5 to 3.5 cm in length as adults. The egg is large (130-150 x 63-90 µm) and has an operculum (clear plug) at one end and appears very different from GIN strongyle-type eggs. While this parasite is common and very destructive in other countries, it is not commonly found in Canada, although a survey in Alberta identified it in sheep, cattle, farmed bison and wapiti (elk). It is important to be alert of its potential occurrence.

The intermediate host is an amphibious snail of the *Lymnaea* genus. The snails prefer wet, low-lying land and so the disease is associated with such pastures. The adult fluke lays eggs in the bile ducts of the liver, which are shed in the faeces. The eggs hatch under warm conditions and produce a miracidium, which must penetrate a suitable snail within 3 h. of hatching. It then divides and develops into as many as 600 cercariae. These cercariae are shed from the snail, and attach themselves to blades of grass where they encyst as metacercariae and are more resistant to the environment. The sheep or goats consume the metacercariae while grazing, which then migrate through the intestinal wall to the liver. The young flukes wander through the liver for about 2 months before moving into the bile ducts where they mature to egg laying adults. The adult flukes may survive for years in the animal. The prepatent period is 10 to 12 weeks.

The disease may be acute, sub-acute or chronic depending on the number of metacercariae ingested and the stage of the disease. If several thousand infect the liver, the damage can be so severe that bleeding and secondary clostridial infections of the liver may occur. Subacute disease is associated with ingestion of smaller numbers of metacercariae (500 to 1500) and disease is evident about 6 to 10 weeks later (late fall, early winter) with bile duct inflammation as well as damage to the liver. The animal has severe anaemia and hypoproteinemia (bottle jaw) and if untreated, will die within 1 to 2 weeks. The chronic form is the most common and is seen in late winter to early spring, 4 to 5 months after ingesting 200 to 500 metacercariae. Again, anaemia and hypoproteinemia are the main presenting signs - but in this case, the fluke eggs can be demonstrated in feces. Sheep and goats do not develop immunity so any age can be affected. Diagnosis can be aided by blood tests that detect evidence of severe liver damage. On postmortem, the liver is enlarged and may be haemorrhagic in the acute form, and scarred and pale in the chronic form with enlarged, thickened bile ducts. The flukes can be seen in the liver and bile ducts.

Regular anthelmintics (fenbendazole, ivermectin) are not effective against flukes. Closantel (Flukiver™, Elanco Canada Ltd) is effective against adults and immature flukes older than 5 weeks, but less effective against younger immature forms. Albendazole (Valbazen®, Zoetis Canada) at double dosage is effective against adult stages only; however, in many countries there is widespread resistance of *F. hepatica* to this anthelmintic. Triclabendazole (Fasinex, Elanco/Novartis), which is effective against all immature stages as well as the adults, is only available in Canada with an emergency drug release, obtained from the Veterinary Drug Directorate, Health Canada. If diagnosed, the flock veterinarian can make a request for importation of this product to treat the animals. Because the intermediate snail host is only found in wet, boggy pastures, fence grazing animals out of these lands.

FASCIOLOIDES MAGNA

This parasite is usually found in deer, wapiti, cattle, and moose, but sheep and goats can be severely affected. It is commonly known as the "**large American liver fluke**" or "**giant liver fluke**". As its name suggests, this is a very large fluke - up to 10 cm (3") in length. The intermediate host is a freshwater snail. The fluke migrates through the liver and causes severe haemorrhage and inflammation. It can penetrate the gut and may cause massive blood loss and infection. They do not enter the bile ducts of the liver and so do not produce eggs in the feces. This infection is common in deer in Ontario and western Canada. Ruminants grazing wet low-lying pastures where deer may also have grazed, have

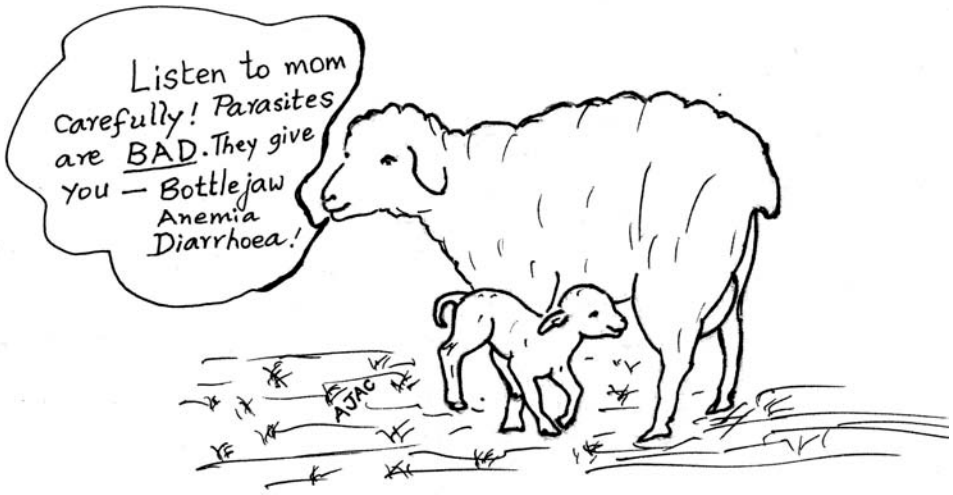
become infected with severe consequences. Since the snail prefers wet, stagnant boggy pastures, it is best to prevent grazing of these kinds of lands. Animals dying on pasture need to have a postmortem performed by a veterinarian. It is sometimes hard to find the flukes if the haemorrhage and infection are severe. The liver should be carefully sliced thoroughly as they may be anywhere in the liver. Treatment of all at risk, healthy appearing animals on a farm is advised. This may prevent further losses. Closantel or triclabendazole can be used when prescribed by your veterinarian.

DICROCOELIUM DENDRITICUM

The final hosts for this liver fluke are sheep, goats, cattle, deer, camelids (llamas and alpacas) and rabbits. It is common in parts of Canada and is also called the "**small lanceolate fluke**". This fluke is very small, < 1 cm and pointed in appearance. The intermediate hosts are firstly a land snail, which produces the cercariae - but then these are ingested by ants. The resulting metacercariae infect the brain of the ant, causing them to act crazy and to climb to the top of blades of grass, where they are more likely to be eaten by grazing ruminants. The adult flukes are very long-lived and infections can be cumulative in bile ducts. The eggs are fluke-type and are smaller than other fluke eggs (38-45 x 22-30 µm). Because of the sylvatic cycle in wildlife, and the fact that land snails are the intermediate host, it may be difficult to avoid infection if present in a geographic location. It is not considered to be very pathogenic to sheep or goats but heavy infections may cause ill thrift. It is more pathogenic to camelids. One case in Ontario has been reported of mortality in sheep associated with copper toxicity, presumably triggered by liver damage from the parasite. There is no parenchymal migration with this fluke. The damage in severe infections is due to bile duct scarring and secondary cirrhosis. The livers are condemned.

GLOSSARY OF ABBREVIATIONS

AAD	Amino-Acetonitrile Derivatives	FECRT	Faecal egg count reduction test
ADG	Average daily gain	GIN	Gastrointestinal nematodes
AR	Anthelmintic resistance	ML	Macrocyclic lactones
BZ	Benzimidazoles	PPER	Periparturient egg rise
CT	Condensed tannins	SL	*Sericae lespedizia*
epg	eggs per gram		
FEC	Faecal egg count		



APPENDICES

1. PROTOCOL FOR COLLECTING FAECAL SAMPLES FOR FAECAL EGG COUNTS

EQUIPMENT AND SUPPLIES:

- Ziploc or sealable sandwich bags
- Ice packs and styrofoam cooler if need to ship to lab
- Disposable gloves and lubricant (if taking samples from the rectum)
- Sharpie black pen (for identifying samples)
- Form on which to record: date samples collected; from which group of animals; from which pasture; age of animals sampled; total number of individual samples collected

SAMPLE COLLECTION

- Collect 8-10g fecal samples (for lambs or kids 10 to 15 fecal pellets and from adults, 6 to 8 fecal pellets) from each of 10 to 15 different animals that are representative of their group (e.g. nursing lambs; weaned kids; pregnant ewes) or pasture.
- Don't mix samples from different groups or pastures.
- The simplest way to collect samples is from the ground. Close the animals in a clean pen or crowd into a clean corner of the pasture, leave them for 15 minutes, release the group and then collect the feces from the ground.
- You can also purposely collect from specific animals. The best way is to put on a glove, use a small amount of lube and using 1 finger in the rectum, tease out the faecal pellets.
- To be sure samples from the ground are fresh; they should be warm and moist. Old samples will give a false negative result as the eggs may have hatched and so won't be visible under the microscope.
- You do not need to keep track of who shed the feces, but if you are interested in individual egg counts, you may do so.

SAMPLE SUBMISSION

Once you have collected the samples in separate Ziploc bags, place them in the Styrofoam boxes, put in the ice-packs, and fill in the records. Deliver the samples to your veterinary clinic while still chilled. If kept cool, the samples are good for a few days but room temperature will allow the eggs to hatch. Forcing as much air as possible out of the Ziploc bag and tightly sealing also helps prevent eggs from hatching. The samples can be processed either by your veterinarian or they may be sent to the Animal Health Laboratory, University of Guelph or other laboratories that offer this service (Ontario producers) for analysis via courier through your veterinarian. Although individual samples are collected, request that samples be "pooled" for analysis; one pooled result per group of animals (e.g. production group, pasture etc).

2. MCMASTER COUNTING TECHNIQUE

PRINCIPLE:

The McMaster counting technique is a quantitative technique to determine the number of eggs present per gram of feces (epg). A flotation fluid is used to separate eggs from fecal material in a counting chamber (McMaster) with two compartments. The technique described below will detect 50 or more epg of feces.

APPLICATION:

This technique can be used to provide a quantitative estimate of egg output for nematodes and coccidia.

EQUIPMENT:

- Beakers or plastic containers
- Balance accurate to 0.1 g
- A tea strainer or cheesecloth
- Measuring cylinder
- Stirring device (tongue depressor)
- Pasteur pipettes and (rubber) teats or plastic pipettes or 1 mL syringe
- Flotation fluid; e.g. salt/sugar solution: 400 g NaCl + 1000 ml water + 500 g sugar (fluid specific gravity = 1.280)
- McMaster slide with 2 counting chambers
- Microscope

PROCEDURE:

- Weigh 4 g of feces and place into Container 1.
- Add 56 ml of flotation fluid.
- Mix (stir) the contents thoroughly with a stirring device (tongue depressor). No lumps should be left.
- Filter the fecal suspension through a tea strainer or a double layer of cheesecloth into Container 2.
- While stirring the filtrate in Container 2, take a sub-sample with a Pasteur pipette or syringe.
- Fill one side of the McMaster counting chamber with the sub-sample. There should be no visible air bubbles.
    - TIP: Moisten the inside of the chambers with water first to reduce the risk of air bubbles forming. Tap/shake out large droplets to avoid diluting the floatation fluid.
- Empty the syringe, stir the filtrate again, and take another sub-sample to fill the other side of the McMaster slide.
- Allow the counting chamber to stand for 5 minutes (this is important).
- Examine the sub-sample of the filtrate under a microscope at 10 x 10 magnification.
- Count all eggs and coccidia oocysts within the engraved area of both chambers. If the numbers in the two chambers are very different, the filtrate was not mixed well and the chambers should be emptied and the count repeated.
- The number of eggs per gram of feces can be calculated as follows: Add the egg counts of the two chambers together. Multiply the total by 50 to give the epg of feces. (Example: 12 eggs seen in chamber 1 and 15 eggs seen in chamber 2 = (12 + 15) x 50 = 1350 epg).

Source: Hansen, J. and Perry, B (1994) The epidemiology, diagnosis and control of helminth parasites of ruminants. International Laboratory for Research on Animal Diseases, P.O. Box 30709, Nairobi, Kenya, pp. 171.

3. RECORD FOR PASTURE USE AND PARASITE CONTROL

5 STAR WORM PLAN PASTURE USE PLANNING SHEET - 20_____

Use this form to track pasture use, results of parasite management of animals. This form is to help you plan pasture use in order to minimize contamination of the pasture with parasite eggs and exposure of livestock to infective parasite larvae.

Pasture	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
FEC												
FAMACHA												
DRENCH												

Record Grazing Details: e.g. species (e.g. sheep, goats, cattle etc), age of animals, number of animals, production stage

Record Pasture Management Details: e.g. resting (no livestock); hay making; seeding; spreading manure; strip grazing

Adapted from A Handbook of Sustainable Worm Management for Livestock Farms

4. RECORD FOR FAECAL EGG COUNT MONITORING

5 STAR WORM PLAN FAECAL EGG COUNT RECORD								
Date Sampled	Pasture	Species / Class	Age / Weight	Date Last Dewormed	RESULTS			Comments
					GIN epg Mean (range)	Coccida opg Mean (range)	Other	

5. ADDITIONAL RESOURCES

ABSTRACTS OF PAPERS ON GASTROINTESTINAL PARASITISM FROM ONTARIO, CANADA

This work, as well as reading other research papers and consulting with international experts, has formed the basis for the handbook. We have attempted to make it as relevant to Canadian sheep and goat producers as possible, while understanding the international context of this disease. Full papers are available on request.

Mederos A, Fernández S, VanLeeuwen J, Peregrine AS, Kelton D, Menzies P, LeBoeuf A, Martin R. ***Prevalence and distribution of gastrointestinal nematodes on 32 organic and conventional commercial sheep farms in Ontario and Quebec, Canada (2006-2008).*** Vet Parasitol. 2010 Jun 24;170(3-4):244-52.

In order to characterize the epidemiology of sheep gastrointestinal nematodes in organic and conventional flocks in Canada, a longitudinal study was carried out from May 2006 to March 2008 on 32 purposively selected farms in Ontario (ON) and Quebec (QC): 8 certified organic (CO), 16 non-certified organic (NCO), and 8 conventional (C) farms. On each farm, 10 ewes and 10 female lambs were selected. Farm visits were undertaken monthly during the grazing season, and twice in the winter. At each visit, individual fecal samples were taken, and pasture samples were obtained during the grazing season. In addition, body condition score was recorded for all sheep. Fecal egg counts per gram of feces (EPGs) were determined for all fecal samples, and infective larvae (L3) were identified in fecal samples (lambs and ewes separately) and pasture samples from farms. Necropsies of 14 lambs from 7 of the 23 Ontario farms were performed at the end of the grazing season in 2006. The mean EPG for year 1 (May 2006 to March 2007) was 181 (range=0-9840) and 351 (range=0-18,940) for the ewes in ON and QC, respectively, and for the lambs was 509 (range=0-25,020) and 147 (range=0-3060) for ON and QC, respectively. During year 2 (April 2007 to March 2008), the mean EPG was 303 (range=0-21,160) and 512 (range=0-22,340) for the ewes in ON and QC, respectively, and for lambs was 460 (range=0-26,180) and 232 (range=0-8280) for ON and QC, respectively. Although the overall mean EPGs were not remarkably high, there were months of higher EPG such as May-June for ewes and July-August for lambs in both provinces. Pasture infectivity was highest in May-June and September. There was a general trend for the CO farms to have lower mean EPG than NCO and C farms. Fecal cultures demonstrated that the most predominant nematode genera were *Teladorsagia* sp., *Haemonchus* sp. and *Trichostrongylus spp.* Pasture infectivity was highest during June-July (984 L3/kg DM) in ON farms and September (mean=436 L3/kg DM) in QC farms during year 1. In year 2, the highest peak was during October in ON (mean=398 L3/kg DM) and July in QC (239 L3/kg DM). *Trichostrongylus axei* and *Trichostrongylus colubriformis* were the species most frequently identified from necropsies (36.44% and 38.26%, respectively) at the end of the grazing season in 2006, with *Haemonchus contortus* and *Teladorsagia circumcincta* being the next most commonly identified.

Guthrie AD, Learmount J, VanLeeuwen J, Peregrine AS, Kelton D, Menzies PI, Fernández S, Martin RC, Mederos A, Taylor MA. ***Evaluation of a British computer model to simulate gastrointestinal nematodes in sheep on Canadian farms.***Vet Parasitol. 2010 Nov 24;174(1-2):92-105.

With increasing levels of anthelmintic resistance worldwide and a growing demand to produce more organic products, utilisation of control strategies for gastrointestinal nematodes (GIN) that minimize the use of anthelmintics becomes even more important. This study evaluated the farm-level performance of an existing predictive sheep parasite model from the United Kingdom (UK), using Canadian data. The UK model simulates the epidemiology of three major GIN species of interest (Teladorsagia spp., Haemonchus spp. and Trichostrongylus spp.) and provides a prediction about seasonal parasite levels of lambs and ewes. Model inputs were generated by using data from the first 2 years of a 3-year study (2006-2008), which examined the epidemiology of GIN parasitism in Ontario sheep flocks. Required input data included ewe parasite egg output, pasture-related information and management dynamics. Farm visits in 2006 and 2007 provided relevant data that were collected monthly during the grazing season, on six and seven occasions respectively. These data were collected from 10 ewes and 10 lambs on each farm. For 23 Ontario farms with available data, only 11 farms in 2006 and 14 in 2007 had suitable data to run in the model because the Canadian study was not specifically designed with this simulation model in mind. Observed values for faecal egg counts (FEC) were compared to the model FEC outputs and assessed using linear regression analysis. There was adequate fit between observed and simulated data for 8 of the 11 farms modelled using data generated in 2006 ($F=7.55-42.66$, $df=10-11$, $R(2)=0.43-0.81$, $p=0.021$ to <0.001) and with 8 of the farms modelled using data generated in 2007 ($F=5.56-35.82$, $df=9-11$, $R(2)=0.36-0.82$, $p=0.040$ to <0.001). We suggest that the poor fit between observed and simulated data for some data sets may be attributable to low-level infection on farms making regression difficult due to insensitivity of the egg count method at low values, or a pattern for immunity in ewes that contradicted the model assumptions. Required model modifications focused on accommodating the differences between UK and Canadian management styles; specifically the practice of bringing lambs indoors for weaning which was sometimes used on Canadian farms.

Mederos A, Waddell L, Sánchez J, Kelton D, Peregrine AS, Menzies P, Vanleeuwen J, Rajić A ***A Systematic Review-Meta-Analysis of Primary Research Investigating the Effect of Selected Alternative Treatments on Gastrointestinal Nematodes in Sheep Under Field Conditions.*** Prev Vet Med. 2012.104:1-14

Selected alternative treatments for preventing or controlling gastrointestinal nematodes (GIN) in sheep under field conditions were evaluated using a systematic review-meta-analysis methodology. Forty-three publications reporting 51 studies (21 controlled studies (CS) and 30 challenge studies (ChS)) and 85 unique treatment comparisons were included in the review. The alternative treatment categories were nutraceuticals (28 studies), breeding for genetic resistance (12), nutritional manipulation (6), homeopathies (2), administration of copper oxide wire particles (2), and biological control (1). Random effect meta-analyses (MA) and meta-regression

were performed with the natural logarithm of the difference in means (lnMD) between the control and treatment groups, for fecal egg counts per gram of wet feces (FEC), worm counts (WC) or fecal egg counts per gram of dry matter (FECDM) as the outcome. Treatment effect estimates (lnMD) were back-transformed to their count ratios (CR), a relative measure of effect for controlled versus treated groups, for presentation of results.

Significant heterogeneity was observed for both CS and ChS that evaluated nutraceuticals, genetic resistance and nutrition treatments. MA of ChS that investigated nutraceuticals resulted in a significant overall CR of 1.62 (P<0.01) and 1.64 (P<0.01) for FEC and FECDM, respectively and a marginal significant CR of 1.14 (P=0.06) for WC, all favoring the treated groups. MA of CS and ChS that investigated genetic resistance resulted in a significant overall CR of 5.89 and 15.42, respectively (P<0.01), again favoring treated groups. MA of CS that investigated homeopathies with FEC as an outcome were homogenous (I(2)=0.0%) and resulted in a non-significant pooled CR of 1.61. ChS investigating copper oxide wire particle treatments and WC as an outcome, were homogenous (I(2)=0.0%) and had a marginally significant pooled CR of 1.68 (P=0.06). Publication bias was observed for ChS with WC outcomes, indicating that small size studies reporting non-significant CR, were less likely to be published than similar studies that found a significant CR. In a meta-regression, randomization (6.2%) and study size (29.2%) were the main factors contributing to the total variation when the outcome was FEC, and none of the variables contributed to between study heterogeneity. When the outcome was WC, type of treatment was the only significant covariate, explaining 6% of the heterogeneity and 38.5% of the total variation. The methodological soundness and reporting of primary research in the selected studies were low. Our results indicate that from the studied alternative treatments, nutraceuticals and use of genetically resistant sheep might be more promising for control of GINs in sheep.

DeWolf BD, Peregrine AS, Jones-Bitton A, Jansen JT, Mactavish J, Menzies PI. ***Distribution of, and risk factors associated with, sheep carcass condemnations due to Cysticercus ovis infection on Canadian sheep farms.*** Vet Parasitol. 2012 Dec 21;190(3-4):434-41.

Cysticercus ovis, the intermediate stage of a canine tapeworm, Taenia ovis, produces cystic lesions in the skeletal and cardiac muscle of sheep, which, if numerous, will result in the condemnation of an entire carcass. In 2007 and 2008, the number of carcass condemnations due to *C. ovis* rose dramatically across Canada, suggesting that the prevalence of this infection on sheep farms was increasing. Trace-back of 237 carcasses condemned at Ontario provincially inspected abattoirs, between March 2009 and March 2011, revealed they originated from 133 farms across Canada. A case-control study was performed (n=40 cases, 56 controls) to identify farm-level risk factors associated with carcass condemnations due to C. ovis. Participating farms, located in Alberta, Saskatchewan, Manitoba and Ontario, were asked to answer a short questionnaire, which collected information about each farm's geographic location and management practices. A multivariable logistic regression model revealed that farm dogs scavenging deadstock (OR=4.04; 95% CI: 1.16-14.04) and failing to dispose of deadstock (OR=11.78; 95% CI: 2.93-47.40) were significantly associated with condemnations ($p \leq 0.05$).

Falzon LC, Menzies PI, Shakya KP, Jones-Bitton A, Vanleeuwen J, Avula J, Stewart H, Jansen JT, Taylor MA, Learmount J, Peregrine AS. ***Anthelmintic resistance in sheep flocks in Ontario, Canada***. Vet Parasitol. 2013 Mar 31;193(1-3):150-62.

Gastrointestinal nematodes (GIN) are a significant constraint to pasture-based sheep production worldwide. Anthelmintic resistance (AR) has been reported in most sheep-raising areas in the world, yet little is known about the AR status in Canada. This study was conducted to determine the frequency of AR in GIN in sheep flocks in Ontario, Canada. Forty-seven sheep flocks were enrolled in the study, and their level of parasitism was monitored monthly throughout a grazing season by analyzing owner-acquired fecal samples from 15 grazing lambs per flock. When the mean GIN fecal egg count (FEC) reached a threshold of 200 eggs per gram (epg), oral ivermectin was supplied to producers to check ivermectin efficacy; the reduction in mean FEC 14 days after ivermectin treatment was calculated. 'Drench failure' was defined as a reduction in mean FEC of <95%. In those flocks with apparent drench failure, researchers performed a Fecal Egg Count Reduction Test (FECRT), dividing sheep into 4 treatment groups (n=10-15): control (i.e. untreated), ivermectin, and, if sufficient numbers of animals - fenbendazole and levamisole. AR was defined as a reduction in mean FEC <95% and a lower 95% confidence interval <90%. Larval cultures were performed on pooled post-treatment FECRT samples. Larval Development Assays (LDAs) to detect the presence of resistance to thiabendazole and levamisole were performed prior to the ivermectin drench check on pooled owner-acquired fecal samples that reached the 200 epg threshold. Approximately 89% (42/47) of the farms reached the FEC threshold of 200 epg; 93% (39/42) of these farms performed an ivermectin drench check, and 88% (34/39) of these farms had drench failure. The FECRT was performed on 29 of the 34 farms. Resistance to ivermectin, fenbendazole and levamisole was demonstrated on 97% (28/29), 95% (19/20) and 6% (1/17) of the farms tested, respectively, with considerable variability in resistance levels among farms. Haemonchus sp. was the most commonly cultured parasite from post-treatment fecal samples. LDA results for 21 farms were available; of these, 14% (3/21) and 62% (13/21) had low and high levels of thiabendazole resistance, respectively, while none of the farms exhibited resistance to levamisole. Amongst these tested farms, resistance to both ivermectin and benzimidazoles was very common. These findings strongly suggest that AR, particularly in Haemonchus sp., is a serious problem in these sheep flocks. Thus, marked changes in GIN management need to be instituted immediately to mitigate a worsening situation.

Falzon LC, Menzies PI, Shakya KP, Jones-Bitton A, Vanleeuwen J, Avula J, Jansen JT, Peregrine AS. ***A longitudinal study on the effect of lambing season on the periparturient egg rise in Ontario sheep flocks.*** Prev Vet Med. 2013 Jul 1;110(3-4):467-80.

The epidemiology of the periparturient egg rise (PPER) of gastrointestinal nematodes (GINs) in sheep remains unclear, and may be influenced by the lambing season. This longitudinal study was performed to determine the effect of out-of-season lambing on the PPER in ewes in Ontario, and whether total plasma protein (TPP) and packed cell volume (PCV) were associated with the PPER. Six farms that practiced out-of-season lambing were enrolled, and sampled for three consecutive lambing seasons (winter, spring and autumn). For each lambing season, all farms were visited five times. On the first visit for each lambing season, 15-20 pregnant ewes and 15-20 non-pregnant/early gestation ewes were randomly selected. At each visit, fecal samples were collected from all selected animals and processed individually to measure GIN fecal egg counts (FECs). Blood samples were collected on three visits in each lambing period and processed to measure TPP and PCV. The ewes were classified into one of five production stages (maintenance [i.e. not pregnant], early or late gestation [<120 d and ≥ 120 d, respectively], and early or late lactation [<40 d and ≥ 40 d, respectively]) based on information collected during farm visits. Linear mixed models were developed for the TPP, PCV and logarithmic-transformed FEC (lnFEC). During the winter and spring lambing season, the FECs increased gradually over the gestation period and peaked during lactation, with these increases being larger in ewes with a low PCV (three-way interaction in the final model). In the autumn lambing season, the FECs started off higher in early gestation, and increased rapidly to peak in late gestation, particularly for animals with low PCV levels. In the TPP model, PCV and lnFEC were positively associated with TPP. During both autumn and winter lambing seasons, the TPP decreased from maintenance throughout gestation and early lactation, followed by an increase in late lactation, except for when there were high FECs. During the spring lambing season, TPP peaked at early gestation, and then decreased in late gestation, to increase more gradually over lactation. In the PCV model, PCV increased with TPP and decreased exponentially with increases in lnFEC. The PPER occurred during all three lambing seasons, and its magnitude and distribution varied with the lambing season, suggesting that the PPER in ewes depends on both environmental and animal physiological factors, an important consideration when implementing preventive parasite control strategies on sheep farms that practice out-of-season lambing.

DeWolf BD, Poljak Z, Peregrine AS, Jones-Bitton A, Jansen JT, Menzies PI. ***Development of a Taenia ovis transmission model and an assessment of control strategies.*** Vet Parasitol. 2013 Nov 15;198(1-2):127-35.

The metacestode stage of the tapeworm, Taenia ovis, causes cystic lesions in the skeletal and cardiac muscle of sheep, which can result in the condemnation of the entire carcass. In recent years, Canadian farms have seen a marked increase in the number of condemnations due to T. ovis. Mathematical transmission models provide a useful tool for predicting parasite transmission and for evaluating the efficacy of potential control options. To date, no model has been developed exclusively for T. ovis. In the work described here, a compartmental, deterministic transmission model was developed to better understand the transmission dynamics of T. ovis on Canadian sheep farms. The model was intended to be practical, and represent the transmission of infection burdens in lambs that result in carcass condemnation, or transmission to canids. All transmission parameters were obtained from the literature or, when unavailable, expert opinion. The model incorporated each stage of the parasite lifecycle using the most probable transmission route on Canadian sheep farms; including definitive host (guard dogs), intermediate host (pastured lambs), and environment. Based on literature, the model performed as expected, and provided a reasonable estimate of parasite prevalence in lambs. In addition, modeling allowed the efficacy of potential control options to be evaluated and compared. Model simulations suggested that infection risk in market lambs could be eliminated through the regular treatment of guardian dogs every fifth week with an appropriate cestocide, or through eliminating carcass consumption by guardian dogs.

Barrere V, Falzon LC, Shakya KP, Menzies PI, Peregrine AS, Prichard RK. ***Assessment of benzimidazole resistance in Haemonchus contortus in sheep flocks in Ontario, Canada: comparison of detection methods for drug resistance.*** Vet Parasitol. 2013 Nov 15;198(1-2):159-65.

In 2011, a field study was conducted to assess drug resistance of gastro-intestinal nematodes in sheep flocks in Ontario, Canada. Benzimidazole resistance in *Haemonchus contortus* was assessed by genetic analysis of eggs; measurement of resistant allele percentages at codons 167, 198 and 200 in the β-tubulin gene was determined on pools of *H. contortus* eggs using pyrosequencing. Susceptibility to benzimidazoles in gastro-intestinal nematodes was also determined using a Faecal Egg Count Reduction Test (FECRT) and a Larval Development Assay (LDA). In total, 16 farms were assessed with the genetic test. Based on resistant allele frequencies, all of the farms (16/16) tested had benzimidazole resistance in *H. contortus*; the overall percentage of benzimidazole-resistant *H. contortus* (estimated prior to treatment using the Hardy-Weinberg formula) was 68.5%. The FECRT and LDA were performed on 11 and 13 farms, respectively. Resistance to fenbendazole was detected on 100% (11/11) of the farms where the FECRT was performed. The LDA revealed the presence of thiabendazole resistance in *H. contortus* in 92% (12/13) of the farms. Estimated percentages of resistant parasites in H. contortus populations obtained with the two biological tests and the genetic test were compared. The results of the genetic test were in agreement with the biological tests and confirmed that benzimidazole resistance in H. contortus is present in Ontario sheep flocks. Differences between the different methods of drug resistance detection are discussed in terms of cost, time and sampling.

De Wolf BD, Peregrine AS, Jones-Bitton A, Jansen JT, Menzies PI. ***Taenia ovis infection and its control: a Canadian perspective.*** N Z Vet J. 2014 Jan;62(1):1-7.

Distributed worldwide, *Taenia ovis* infection is responsible for the condemnation of sheep carcasses in many countries. This review highlights the programme used in New Zealand to successfully control *T. ovis* in sheep, and discusses how similar approaches may be modified for use in Canada, given what is currently known about the epidemiology of *T. ovis*. The lifecycle of the parasite is well known, involving dogs as the definitive host and sheep or goats as the intermediate host. An effective vaccine does exist, although it is not presently commercially available. In New Zealand an industry-based, non-regulatory programme was created to educate producers about T. ovis and necessary control strategies, including the need to treat farm dogs with cestocides regularly. This programme resulted in a substantial decrease in the prevalence of T. ovis infections between 1991 and 2012. Historically, T. ovis was not a concern for the Canadian sheep industry, but more recently the percentage of lamb condemnations due to T. ovis has increased from 1.5% in 2006 to 55% in 2012. It has been suggested that coyotes may be transmitting T. ovis, but this has not been confirmed. Recommendations are made for the Canadian sheep industry to adopt a control programme similar to that used in New Zealand in order to reduce the prevalence of T. ovis infection.

Falzon LC, Menzies PI, VanLeeuwen J, Shakya KP, Jones-Bitton A, Avula J, Jansen JT, Peregrine AS. ***Pilot project to investigate over-wintering of free-living gastrointestinal nematode larvae of sheep in Ontario, Canada.*** Can Vet J. 2014 Aug;55(8):749-56.

This study investigated the overwintering survival and infectivity of free-living gastrointestinal nematode (GIN) stages on pasture. The presence of GIN larvae was assessed on 3 sheep farms in Ontario with a reported history of clinical haemonchosis, by collecting monthly pasture samples over the winter months of 2009/2010. The infectivity of GIN larvae on spring pastures was evaluated using 16 tracer lambs. Air and soil temperature and moisture were recorded hourly. Free-living stages of *Trichostrongylus spp.* and *Nematodirus spp.* were isolated from herbage samples. Gastrointestinal nematodes were recovered from all tracer lambs on all farms; *Teladorsagia* sp. was the predominant species. Very low levels of *Haemonchus contortus* were recovered from 1 animal on 1 farm. The results suggest that *Haemonchus* larvae do not survive well on pasture, while *Teladorsagia sp., Trichostrongylus spp. and Nematodirus spp.* are able to overwinter on pasture in Ontario and are still infective for sheep in the spring.

Falzon LC, O'Neill TJ, Menzies PI, Peregrine AS, Jones-Bitton A, VanLeeuwen J, Mederos A. ***A systematic review and meta-analysis of factors associated with anthelmintic resistance in sheep.*** Prev Vet Med. 2014 Nov 15;117(2):388-402.

BACKGROUND: Anthelmintic drugs have been widely used in sheep as a cost-effective means for gastro-intestinal nematode (GIN) control. However, growing anthelmintic resistance (AHR) has created a compelling need to identify evidence-based management recommendations that reduce the risk of further development and impact of AHR.

OBJECTIVE: To identify, critically assess, and synthesize available data from primary research on factors associated with AHR in sheep.

METHODS: Publications reporting original observational or experimental research on selected factors associated with AHR in sheep GINs and published after 1974, were identified through two processes. Three electronic databases (PubMed, Agricola, CAB) and Web of Science (a collection of databases) were searched for potentially relevant publications. Additional publications were identified through consultation with experts, manual search of references of included publications and conference proceedings, and information solicited from small ruminant practitioner list-serves. Two independent investigators screened abstracts for relevance. Relevant publications were assessed for risk of systematic bias. Where sufficient data were available, random-effects Meta-Analyses (MAs) were performed to estimate the pooled Odds Ratio (OR) and 95% Confidence Intervals (CIs) of AHR for factors reported in ≥2 publications.

RESULTS: Of the 1712 abstracts screened for eligibility, 131 were deemed relevant for full publication review. Thirty publications describing 25 individual studies (15 observational studies, 7 challenge trials, and 3 controlled trials) were included in the qualitative synthesis and assessed for systematic bias. Unclear (i.e. not reported, or unable to assess) or high risk of selection bias and confounding bias was found in 93% (14/15) and 60% (9/15) of the observational studies, respectively, while unclear risk of selection bias was identified in all of the trials. Ten independent studies were included in the quantitative synthesis, and MAs were performed for five factors. Only high frequency of treatment was a significant risk factor (OR=4.39; 95% CI=1.59, 12.14), while the remaining 4 variables were marginally significant: mixed-species grazing (OR=1.63; 95% CI=0.66, 4.07); flock size (OR=1.02; 95% CI=0.97, 1.07); use of long-acting drug formulations (OR=2.85; 95% CI=0.79, 10.24); and drench-and-shift pasture management (OR=4.08; 95% CI=0.75, 22.16).

CONCLUSIONS: While there is abundant literature on the topic of AHR in sheep GINs, few studies have explicitly investigated the association between putative risk or protective factors and AHR. Consequently, several of the current recommendations on parasite management are not evidence-based. Moreover, many of the studies included in this review had a high or unclear risk of systematic bias, highlighting the need to improve study design and/or reporting of future research carried out in this field.

Mederos A, Kelton D, Peregrine AS, VanLeeuwen J, Fernández S, LeBoeuf A, Menzies P, Martin R. ***Evaluation of the utility of subjective clinical parameters for estimating fecal egg counts and packed cell volume in Canadian sheep flocks.*** Vet Parasitol. 2014 Oct 15;2015(3-4):568-74.

A study was conducted in sheep on Canadian farms to describe the relationship between packed cell volume (PCV) or fecal egg counts (FEC) and subjective clinical parameters that may indicate the severity of parasitic gastroenteritis. Twenty-one farms in Ontario (ON) and 8 farms in Quebec (QC) were purposively selected and visited during April-May (spring) and August (summer) 2007. At each farm visit, blood and fecal samples were collected from 10 ewes and 10 female lambs; body condition score (BCS), dag score (DS), fecal consistency score (FCS) and FAMACHA score were recorded for all sampled sheep. Packed cell volume was determined for all blood samples, and FEC were performed for all fecal samples. Summary statistics and simple correlations were performed for the parameters

recorded. Two mixed models with random effects at the farm level were developed; one using PCV as the response variable and another using the natural log of eggs per gram of feces (lnEPG). Finally, the residuals from both models were correlated to the covariates in the models. The mean PCV values during the spring were 29.7% and 36.7% for lambs, and 28.8% and 31.1% for ewes, in ON and QC, respectively. During the summer, the mean PCV was 32.0% and 32.8% for lambs, and 30.1% and 29.9% for ewes, in ON and QC, respectively. The arithmetic mean FEC per gram of feces (EPG) during the spring was 3 and 2 for lambs, and 1266 and 789 for ewes, in ON and QC, respectively, whereas during summer the arithmetic mean EPG was 907 and 237 for lambs, and 458 and 246 for ewes, in ON and QC, respectively. Results from simple correlations indicated that PCV was negatively correlated with lnEPG (r = -0.255; r(2) = 6.5%) and FAMACHA (r = -0.312; r(2) = 9.7%), and positively correlated with BCS (r = 0.317; r(2) = 10%). LnEPG was negatively correlated with BCS (r = -0.232; r(2) = 5.4%) and PCV (r = -0.255; r(2) = 6.5%), but positively correlated with FAMACHA (r = 0.178; r(2) = 3.2%) and DS (r = 0.086; r(2) = 0.7%). Results from the models indicated that PCV and lnEPG residuals were negatively correlated with FAMACHA, FCS and almost all categories of BCS and DS, although the correlations were very low. The main results from this study suggested that none of the subjective clinical parameters evaluated were highly correlated with PCV or lnEPG and therefore were not good predictors of lnEPG or PCV on the studied farms in Ontario and Quebec.

Falzon LC, van Leeuwen J, Menzies PI, Jones-Bitton A, Sears W, Jansen JT, Peregrine AS. ***Comparison of calculation methods used for the determination of anthelmintic resistance in sheep in a temperate continental climate.*** Parasitol Res. 2015 Apr;114(4):1631-43..

This study compared results obtained with five different fecal egg count reduction (FECR) calculation methods for defining resistance to ivermectin, fenbendazole, and levamisole in gastrointestinal nematodes of sheep in a temperate continental climate: FECR1 and FECR2 used pre-and post-treatment fecal egg count (FEC) means from both treated and control animals, but FECR1 used arithmetic means, whereas FECR2 used geometric means; FECR3 used arithmetic means for pre- and posttreatment FECs from treated animals only; FECR4 was calculated using only arithmetic means for post-treatment FECs from treated and control animals; and FECR5 was calculated using mean FEC estimates from a general linear mixed model. The classification of farm anthelmintic resistance (AR) status varied, depending on which FECR calculation method was used and whether a bias correction term (BCT, i.e., half the minimum detection limit) was added to the zeroes or not. Overall, agreement between all methods was higher when a BCT was used, particularly when levels of resistance were low. FECR4 showed the highest agreement with all the other FECR methods. We therefore recommend that small ruminant clinicians use the FECR4 formula with a BCT for AR determination, as this would reduce the cost of the FECRT, while still minimizing bias and allowing for comparisons between different farms. For researchers, we recommend the use of FECR1 or FECR2, as the inclusion of both pre- and posttreatment FECs and use of randomly allocated animals in treatment and control groups makes these methods mathematically more likely to estimate the true anthelmintic efficacy.

Westers T, Jones-Bitton A, Menzies P, Van Leeuwen J, Poljak Z, Peregrine AS. ***Efficacy of closantel against ivermectin- and fenbendazole-resistant Haemonchus sp. in sheep in Ontario, Canada.*** Vet Parasitol. 2016 Sep 15;228:30-41.

In Ontario, Canada, widespread resistance to ivermectin and fenbendazole, the only readily available ovine anthelmintics, has been documented, primarily in Haemonchus sp. In other parts of the world, closantel has been used to control such infections; however, the drug was not currently licensed for use in Canada and the USA. A randomized controlled trial was conducted on six client-owned farms in Ontario in 2013 and 2014 to determine the efficacy of closantel (Flukiver® 5% Oral Suspension, Elanco Animal Health, 10mg/kg bodyweight) against ivermectin- and fenbendazole-resistant Haemonchus sp. infections in periparturient ewes and grazing lambs. Three farms were randomly assigned to treat all ewes, and three farms were randomly assigned to selectively treat individual ewes at lambing, using predetermined criteria. Fecal samples were collected from a minimum of 15 randomly selected ewes and 13 lambs per group on each farm at the time of treatment and approximately 14days later. Trichostrongyle-type fecal egg counts (FEC) were performed using a modified McMaster technique with a lower detection limit of 8.3 eggs per gram of feces (epg). Haemonchus-specific FECs were determined by multiplying FECs by the proportion of Haemonchus sp. identified from coproculture for each farm; Haemonchus-specific FEC reductions were calculated for each farm. Twenty grazing lambs had FECs conducted monthly, and when mean monthly FECs surpassed 200 epg, all lambs were randomly allocated to either closantel, positive control (ivermectin, fenbendazole, or levamisole) or negative control groups. Pre-treatment Haemonchus-specific mean FECs ranged from 27 to 3359 epg in ewes and 0-5698 epg in lambs. Efficacy of closantel against Haemonchus sp. ranged from 99% (95% CI: 97%-99%) to 100% in recently lambed ewes on all farms in both years (total n=274 ewes), and from 99% (95% CI: 98%-99%) to 100% in grazing lambs in both years on all but one farm (total n=171 lambs). On the latter farm, a whole flock treated farm, closantel efficacy in grazing lambs was 84% (95%CI: 81%-88%) in the first year, but 100% in the second year. Levamisole was effective against overall GIN in lambs on only two farms. Ivermectin and fenbendazole resistance continued to be present, particularly in Haemonchus sp. Closantel had excellent efficacy against Haemonchus sp. over the two year study period, regardless of treatment group, and therefore should be considered one viable component of sustainable integrated parasite control programs for farms with documented anthelmintic resistance and problems with haemonchosis.

Westers T, Jones-Bitton A, Menzies P, VanLeeuwen J, Poljak Z, Peregrine AS. ***Identification of effective treatment criteria for use in targeted selective treatment programs to control haemonchosis in periparturient ewes in Ontario, Canada.*** Prev Vet Med. 2016 Nov 1;134:49-57.

Haemonchosis is often associated with late gestation and parturition in ewes in Canada. Due to widespread concerns about development of anthelmintic resistance (AR), targeted selective treatment (TST), where individual animals are treated with an anthelmintic rather than the entire flock, is a possible strategy to control clinical signs in recently lambed ewes while still maintaining parasite refugia. Performing fecal egg counts (FEC) on individual animals is often cost-prohibitive, so indicators that identify ewes with high FEC are essential for TST programs. The study objectives were to: a) evaluate the ability of four TST indicators to identify periparturient ewes with high Haemonchus sp. FEC and b) determine appropriate treatment thresholds for statistically-significant indicators. A field study was conducted during the 2013 and 2014 lambing seasons (February-May) on three client-owned farms in Ontario with documented AR and problems with haemonchosis in ewes. Ewes were examined within three days of lambing and selected for treatment with oral closantel (10mg/kg body weight), a novel anthelmintic to Canada, if they met at least one of four criteria: a) the last grazing season was their first grazing season; b) body condition score ≤2; c) Faffa Malan Chart (FAMACHA©) score ≥3; and/or d) three or more nursing lambs. Fecal samples were collected per rectum on the treatment day from each of 20 randomly selected treated and untreated ewes on each farm. Haemonchus sp. percentages on each farm, as determined by coproculture, ranged from 53% to 92% of total fecal trichostrongyle-type egg counts. Mean Haemonchus sp. FECs were significantly higher in treated ewes (n=136) than in untreated ewes (n=103) on the day of treatment in both years (p=0.001), suggesting the indicators were suitable for identifying animals with high Haemonchus sp. FEC. A linear mixed model was fit with logarithmic-transformed Haemonchus sp. FEC as the outcome variable, the four indicators and year as fixed effects, and farm as a random effect. FAMACHA© score was the sole indicator to remain significantly associated with FEC (p=0.002). A receiver-operator curve determined that test sensitivity was maximized (92.4%) with FAMACHA© score ≥3 as the sole indicator. FAMACHA© score should therefore be included in TST programs to identify ewes requiring treatment at lambing due to Haemonchus sp.

BOOKS AND TECHNICAL MANUALS

Sheep Flock Health - a planned approach. Neil Sargison. Blackwell Publishing, Oxford UK. 2008.

Smart Drenching and FAMACHA©, Integrated Training for Sustainable Control of Gastrointestinal Nematodes in Small Ruminants. Southern Consortium for Small Ruminant Parasite Control.

Sustainable Worm Control Strategies for Sheep, a technical manual for veterinary surgeons and advisors 3rd edition. K.A. Abbott, M. Taylor, L.A. Stubbings editors. April 2009.

Veterinary Parasitology, 3rd edition.M.A. Taylor, R. L. Coop and R. L. Wall editors.Blackwell Publishing, Oxford UK. 2007.

The epidemiology, diagnosis and control of helminth parasites of ruminants. Hansen J, Perry B. ILRAD 1994. FAO. http://www.fao.org/wairdocs/ilri/x5492e/x5492e00.htm#Contents

WEB-BASED VIDEOS AND INFORMATION

Wormboss. Australia's sheep and goat worm control measures. http://www.wormboss.com.au

Paraboss. for professionals. Australia. https://www.paraboss.com.au

SCOPS (Sustainable Control of Parasites) United Kingdom. http://www.scops.org.uk

American Consortium for Small Ruminant Parasite Control. https://www.wormx.info

War of the Worms. Moredun Research Institute. https://www.youtube.com/watch?v=nV7ItbKCKhE

FAMACHA Score Your Meat Goat. Purdue Extension. https://www.youtube.com/watch?v=BAdeVez5yyc&feature=related

Online FAMACHA© Certification. University of Rhode Island. https://web.uri.edu/sheepngoat/famacha/

Sheep Fecal Sampling Video. University of Guelph. https://www.youtube.com/watch?v=X2C9sVstce0&feature=relmfu

Turn your smart phone into a digital microscope. Kenji Yoshino. https://www.youtube.com/watch?v=KpMTkr_aiYU

CarLA Saliva Test. Agresearch NZ. https://www.agresearch.co.nz/doing-business/products-and-services/carla-saliva-test/

FDA's public meeting on antiparasitic drug use and resistance in ruminants and equines. Food and Drug Administration, USA. https://www.fda.gov/AnimalVeterinary/ResourcesforYou/ucm318015.htm

Guide for the control of external parasites of sheep and goats. New Mexico State University. http://aces.nmsu.edu/pubs/_b/B112/

Liceboss. Australia. http://www.liceboss.com.au

NADIS Animal Health Skills Parasite Forecast (model based on weather) http://www.nadis.org.uk/parasite-forecast/

Ovis Management Ltd (New Zealand) https://www.sheepmeasles.co.nz

SELECTED RESEARCH AND REVIEW PAPERS

Besier RB, Kahn LP, Sargison ND, Van Wyk JA. Diagnosis, treatment and management of *Haemonchus contortus* in small ruminants. Adv Parasitol. 2016;93:181-238.

Charlier J, van der Voort M, Kenyon F, Skuce P, Vercruysse J. Chasing helminths and their economic impact on farmed ruminants. Trends Parasitol. 2014 Jul;30(7):361-7.

Coles GC, Bauer C, Borgsteede FHM et al. World Association for the Advancement of Veterinary Parasitology (W.A.A.V.P.) methods for the detection of anthelmintic resistance in nematodes of veterinary importance. Vet Parasitol 1992;44:35-44

Craig TM. Gastrointestinal nematodes, diagnosis and control. Vet Clin North Am Food Anim Pract. 2018 Mar;34(1):185-199.

Emery DL, Hunt PW, Le Jambre LF. *Haemonchus contortus*: the then and now, and where to from here? Int J Parasitol. 2016 Nov;46(12):755-769.

Eysker M, Bakker N, Kooyman FNJ et al. The possibilities and limitations of evasive grazing as a control measure for parasitic gastroenteritis in small ruminants in temperate climates. Vet Parasitol 2005;129:95-105.

Fleming SA, Craig T, Kaplan RM et al. Anthelmintic resistance of gastrointestinal parasites in small ruminants. J Vet Intern Med 2006;20:435B444

Houdijk JGM, Kyriazakis I, Jackson F et al. Effects of protein supply and reproductive status on local and systemic immune responses to *Teladorsagiacircumcincta* in sheep. Vet Parasitol 2005;129:105B117.

Hoste H, Torres-Acosta JF, Quijada J, Chan-Perez I, Dakheel MM, Kommuru DS, Mueller-Harvey I, Terrill TH. Interactions between nutrition and infections with *Haemonchus contortus* and related gastrointestinal nematodes in small ruminants. Adv Parasitol. 2016;93:239-351.

Kenyon F, Greer AW, Coles GC et al. The role of targeted selective treatments in the development of refugia-based approaches to the control of gastrointestinal nematodes of small ruminants. Vet Parasitol 2009;164:3-11.

Keeton STN, Navarre CB. Coccidiosis in Large and Small Ruminants. Vet Clin North Am Food Anim Pract. 2018 Mar;34(1):201-208.

Kotze AC, Prichard RK. Anthelmintic Resistance in Haemonchus contortus: History, Mechanisms and Diagnosis. Adv Parasitol. 2016;93:397-428.

Langrová I, Makovcová K, Vadlejch J et al. Arrested development of sheep strongyles: onset and resumption under field conditions of Central Europe. Parasitol Res 2008;103:387B392

Larsen M. Biological control of nematode parasites in sheep. J AnimSci 2006;84(E. Suppl.):E133-E139

Learmount J, Taylor MA, Smith G et al.A computer model to simulate control of parasitic gastroenteritis in sheep on UK farms. Vet Parasitol 2006;142:312B329.

LeJambre LF, Windon RG, Smith WD. Vaccination against *Haemonchuscontortus*: performance of native parasite gut membrane glycoproteins in Merino lambs grazing contaminated pasture. Vet Parasitol 2008;153:302-312.

Louie K, Vlassoff A, Mackay AD. Gastrointestinal nematode parasites of sheep: A dynamic model for their effect on liveweight gain. Int J Parasitol 2007;37:233-241.

Marley CL, Fraser MD, Fychan R et al. Effect of forage legumes and anthelmintic treatment on the performance, nutritional status and nematode parasites of grazing lambs. Vet Parasitol 2005;131:267B282

Matthews JB, Geldhof P, Tzelos T, Claerebout E. Progress in the development of subunit vaccines for gastrointestinal nematodes of ruminants. Parasite Immunol. 2016 Dec;38(12):744-753.

Mavrot F, Hertzberg H, Torgerson P. Effect of gastro-intestinal nematode infection on sheep performance: a systematic review and meta-analysis. Parasit Vectors. 2015 Oct 24;8:557.

Miller JE, Horohov DW. Immunological aspects of nematode parasite control in sheep. J AnimSci 2006;84(E. Suppl.):E124-E132.

Min BR, Barry TN, Attwood GT et al. The effect of condensed tannins on the nutrition and health of ruminants fed fresh temperate forages: a review. Anim Feed Sci Tech. 2003;106:3-19.

Morgan ER, Cavill L, Curry GE et al. Effects of aggregation and sample size on composite faecal egg counts in sheep. Vet Parasitol 2005;131:79B87.

O'Connor L, Walkden-Brown SW, Kahn LP. Ecology of the free-living stages of major trichostrongylid parasites of sheep. Vet Parasitol 2006;142:1-15.

Pasture Production. Ministry of Agriculture, Food and Rural Affairs. Order Publication 19, Agdex #130. http://www.omafra.gov.on.ca/english/crops/pub19/pub19toc.htm

Pollott GE, Karlsson LJE, Eady S et al. Genetic parameters for indicators of host resistance to parasites from weaning to hogget age in Merino sheep. J AnimSci 2004;82:2852B2864.

Sweeney T, Hanrahan JP, Ryan MT, Good B. Immunogenomics of gastrointestinal nematode infection in ruminants - breeding for resistance to produce food sustainably and safely. Parasite Immunol. 2016 Sep;38(9):569-86.

vanWyk JA, Bath GF. The FAMACHA system for managing haemonchosis in sheep and goats by clinically identifying individual animals for treatment. Vet Res 2002;33:509B529.

Zajac AM. Gastrointestinal nematodes of small ruminants: life cycle, anthelmintics, and diagnosis. Vet Clin Food Anim. 2006;22:529-541.

Zvinorova PI, Halimani TE, Muchadeyi FC, Matika O, Riggio V, Dzama K. Breeding for resistance to gastrointestinal nematodes - the potential in low-input/output small ruminant production systems. Vet Parasitol. 2016 Jul 30;225:19-28.